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Original Contributions.

ON CASES OF HÆMORRHAGE INTO THE EYE OCCURRING
IN YOUNG MEN.

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I PUBLISHED in the first volume of the Ophthalmological Transactions, 1881, the case of a young man, Colin P., who had suffered from repeated attacks of hæmorrhages into the eyes. The notes of his case extended over five years, and one eyeball had been excised, on account of glaucomatous tension and pain following hæmorrhage into the vitreous, two years before he came under my care. He had, during the two years he had remained under my observation, repeated hæmorrhages into the vitreous of his remaining eye, which used to make him for a time all but blind. My notes included a report by Dr. Mahomed as to the condition of arterial tension, which was found below normal, and I took occasion to refer to an important paper by Mr. Eales, of Birmingham, in which five cases had been recorded, four of them very similar to my own. Mr. Eales had attached great importance to the existence of constipation as a cause of the disturbance of the balance of circulation, and a predisponent to hæmorrhage. In my own case I was inclined to attach much importance to a strong

family history of gout; my patient suffered extremely from dyspepsia, and had himself had pains which were probably of a gouty character. His case quite coincided with Mr. Eales' experience that constipation and epistaxis are usually concomitants with intra-ocular hæmorrhage, and that the subjects of these cases are almost invariably young men. I revert to the subject on the present occasion because I am now able to record, after an eight years interval, a statement as to the present condition of my patient. It is very desirable indeed that we should get completed histories in such cases, for they then become invaluable for purposes of prognosis.

Additional Note of Case of Hæmorrhage into Eye.

Colin Perrot, æt. 27, came to me again May 1885. He has been living in Devonshire ever since I saw him, helping in a grocer's shop. He has read the paper every day. Has had no relapses whatever. The cloud has not wholly disappeared. He can see the cloud on both sides when he looks over his nose, and when he looks to his temple. He sees $\frac{20}{20}$ with ease, and reads No. 1. He has taken neither beer nor wine since I saw him, but occasionally a little spirit. He has been comparatively free from cold feet and has had less rheumatic pain. I do not think that his pulse indicates any increase of tension. It is rather sharp, but not difficult of compression. He has had constipation as much as ever, and has found no benefit from green vegetables. No bleeding from nose. Emissions very rarely—once in two or three months. His head is clear; very seldom headache; appetite always good. Heart's sounds clear and sharp. He complains much of attacks of discomfort at his heart, and has a sense of soreness to left of nipple.

These attacks are not usually attended by palpitation. They consist rather in a sense of fulness. When very bad he breaks out suddenly into profuse perspiration.

He cannot lift with his left arm on account of the discomfort caused at his heart.

He has dieted most carefully.

He thinks that weather influences him much. When it is clear he is better.

Colin P. wrote to me again in 1888 from Devonshire, informing me that he had, since I last saw him, much improved in health, and that he had ceased to be liable to the attacks of bleeding. His eye, which had been so repeatedly and so severely affected, had of late years served him well, and he could follow his occupation and also read in moderation.

This result confirms my previous impression that the liability to intra-ocular hæmorrhage of this kind is closely connected with the age of the patient, and very probably with the disturbing influence of the sexual system during the early years of manhood. It occurs at the same time and under the same conditions that the liability to severe acne of the common type is often noticed, and it would appear to cease at the same time. In saying this, I by no means ignore the influence of other causes, such as inherited gout and neglected constipation. Age and sex are, however, clearly, I think, by far the most important predisponents. It is very seldom, indeed, that we hear of cases of recurring intra-ocular hæmorrhage in young women. In the female sex the occurrence of menstruation saves from risk in this direction. That it may not do so absolutely is very probable; but I believe it will be a matter of general experience that not only is intra-ocular hæmorrhage almost unknown in young women, but that epistaxis is far less troublesome and less severe than it is in males. The subject is one of great interest, and has not as yet attracted much attention. I am not aware that anything has been written upon it since the papers by Mr. Eales and myself. It, therefore, seems worth while to place on record a few other cases which I have since found in my note-books. I might have recorded them at the time of my former paper, had they not been overlooked, for they were written out, and were the subject of a clinical lecture delivered at Moorfields in 1871. The fact that they were observed

prior to my former paper must be my excuse for not having noted in more detail the histories of the patients as regards gout, and in reference to arterial tension, &c. Both the patients with recurrent hæmorrhages were, according to rule, young males, but I have notes of several cases in which single attacks of hæmorrhage occurred in patients of middle age, and two in which the subjects of it were women. These latter cases, though they must not be lost sight of in connection with the subject, must probably be assigned to a somewhat different clinical category. In all the recurrent cases, both eyes were at one time or another attacked, whereas in these patients at middle age, not only was there no proof of tendency to recurrence, but, so far as my notes go, only one eye was affected. When we get to senile periods of life, and in connection with senile degeneration of arteries, &c., we again encounter attacks of hæmorrhage into the vitreous, and sometimes in association with liability to epistaxis. In these cases, however, the attacks are for the most part single, affecting only one eye, and, as it were, accidental, whereas in the group of cases to which I am now asking attention, the tendency to recurrence during several years is the most marked feature.

In close connection with these cases of recurrent hæmorrhage into the vitreous are yet more rare cases of hæmorrhagic choroiditis. A very marked example of this disease, occurring in a man named Henry Gorham, who was under my care in 1874, is recorded in the paper to which reference has been made. It occurred at the same age and under precisely similar conditions to those in which the vitreous was the seat of the disease, the patient being the subject of marked sexual disturbance and liable to epistaxis.*

The liability to retinal detachment, which is unfortunately observed in a considerable proportion of the cases of the kind referred to, is probably to be explained by the occasional occurrence of sub-retinal, and possibly of choroidal, bleeding. This is illustrated in my paper in

* See page 35, vol. i, Transactions of the Ophthalmological Society.

the case of a lad named Sullivan, aged 19, who was left quite and permanently blind by this occurrence.

Extensive spontaneous Hæmorrhage into the Vitreous (its front part) in both Eyes in a Young Man liable to severe Epistaxis. Recovery of excellent sight. History of several previous attacks. No cause assignable.

(F. 406.) Thos. Rogers, a spare, healthy-looking young man of 20, was admitted at Moorfields on November 2, 1871, with large hæmorrhages into the vitreous in both eyes; the blood could easily be seen by oblique illumination, and some of it was of a very bright red colour. The optic disc could be just seen in the left eye, and below it (real position) was a vertical line, the nature of which was doubtful, but apparently depending on some change in the retina.

He was taken into the Hospital, and remained an in-patient for seven weeks. Sight improved considerably during his stay, and continued to progress after he returned home. In October, 1872, ten months after his discharge from Moorfields, he wrote that he had "quite recovered his sight." The treatment during his stay in hospital consisted of the solution of bichloride of mercury in drachm doses, three times a day, for five weeks, and iodide of potassium (5-grain doses) for the remaining time. While taking the bichloride he suffered more than once from severe frontal and temporal headache, which kept him awake. He had no more pain after the change of medicine.

The history was that during the twelve months previous to admission he had had several attacks of dimness of sight, sometimes in one eye, sometimes in the other, and seldom in both at once. The dimness came on rapidly, "it would come like a lot of black specks, as I was eating my dinner;" it usually passed off in two or three weeks, and he would again have good sight for a couple of months or so. It was for the most recent attack that he came to the Hospital, its greater severity and longer duration making him anxious, especially as both eyes were affected. His account was that he had been out in camp with the Volunteers at Aldershot in September, had returned home, seeming quite well, one Saturday evening, and that on the Sunday

morning when he awoke his sight was as bad as on admission to the Hospital on November 2.

In respect to the cause of his intra-ocular bleeding, some facts of interest were obtained. He said that for about the last four years he had been liable to repeated and severe epistaxis: his nose would begin to bleed without apparent cause, and even sometimes as he was walking; "sometimes he thought it was never going to stop." This tendency had ceased at about the time he became liable to the attacks of dimness of sight, and for the last 12 months his nose had scarcely bled at all. He denied masturbation, and would not admit ever having had intercourse; his manner was truthful. He had on one occasion, a year and a half ago, been laid up for three months in hospital by a cough, and nearly a year after I saw him he wrote that his breath troubled him much, and that he was taking cod-liver oil under advice. It is probable therefore that he is phthisical. With regard to the effect of straining in causing hæmorrhage, he said that coughing had never brought on epistaxis, and that he had no cough when the present attack of dimness began; and further that when once in hospital with cough, he was constipated for a week, but that he had no special epistaxis then nor any attacks of dimness. He was not aware that any relatives had been liable to bleed unusually, nor did he himself bleed more than other people from cuts on the fingers, &c.

Repeated large Hæmorrhages into the Vitreous of both Eyes with Œdema of Retina and Swelling of Optic Disc. Subsequent Detachment of Retina in one Eye. Patient, a somewhat delicate youth, subject to Epistaxis. No cause assigned for the Eye disease.

(F. 277.) Frank Trickett, æt. 17, a clerk, came to me at Moorfields in April, 1871, on account of recent failure of sight in his left eye. He said that nine months previously (July, 1870) the right failed, and that a well-known oculist whom he consulted said he had "apoplexy in the eye." Three months after that the left became affected; it improved and remained well till three days before I saw him, when the sight rapidly failed again. The right also had almost recovered.

When admitted, he could with the *right* read 1 J. and $\frac{20}{40}$ Sn.

With the *left* he was nearly blind; the disc was swollen and hazy, the veins large and tortuous; it was noted that there were no extravasations, but the existence of thrombi was suspected. There is no note of any opacities in the vitreous. In the right, some floating webs were seen in the vitreous, and the edge of the disc was indistinct; refraction hypermetropic. Small doses of iodide of potassium were ordered.

About two months later, he appears to have had a recurrence of bleeding in both eyes, for on July 6 in the left eye "abundance of blood can be seen by oblique illumination in the lower and lateral parts of the vitreous," completely obscuring the choroid; the choroidal reflex could be obtained only from the upper part. The blood was dark-red, and some of it was in sufficiently thin layers to be translucent. In the right there were many floating opacities, while at the lower part focal light showed the vitreous to be of greyish-red colour.

October 19. "Has been away in the country for a long time, and his health has improved. Sight remained about the same till a week ago (a few days after his return to London), when the *right* again became much worse, and he can now only just see his way about." In the *right* we found a large retinal detachment at the lower and outer part, and many floating bodies in the vitreous; in the *left* the blood had become to a great extent decolourised, and there was a whitish reflection from all parts which might be due entirely to the condition of the vitreous, or be in part caused by detachment of the retina. He could not read the largest letters with either.

November 30. With the *right* he can now see 8 J.; fundus very hazy, and disc cannot be seen (atropine not used). The improvement was remarkable. I have not seen him since.

This patient was a pale lad with fair hair and tolerably well nourished; he said that he had never been considered robust, but his muscular strength was good, and he was able to walk long distances. From his earliest remembrance he had been liable to epistaxis, especially in hot weather. He believed that he had scarlet fever when about 10 years of age, but had had no dropsy nor any rheumatic affection. There seemed no reason for doubting his

assertion that he was not given to masturbation. His epistaxis, although troublesome, does not appear ever to have been excessive, and while he was under observation for his eye-symptoms it was, he stated, less frequent than it had formerly been.

In December, 1875, I again heard from him. After leaving, the hospital in November, 1871, he was under care elsewhere, and in six months "was able to see clearly." He continued to retain good sight and improved health till the present autumn, when his health again failed somewhat, and he "lost the use of his left eye again, the retina being covered with blood," to use his own expression. He was already feeling better, and the eye had again begun to improve.

Spontaneous Hæmorrhages into the Vitreous (Symmetrical but not Synchronous) in a Patient subject to various other Bleedings from slight causes or without cause. Family history of Hæmorrhagic Diathesis.

Robert Beard, 22, carpenter (formerly Mr. Hulke's patient). in-patient November, 1871; again November, 1872.

Fair hair and clear rosy complexion; grey eyes. Tall and rather thin. Skin smooth, not hairy.

In August, 1871, L. eye became gradually dim; in about three weeks could only see shadows. End of November (three to four months) it recovered, so that he could read books, &c., but only for short time at once. At end of November R. was attacked, but much more suddenly; in a few hours could only see shadows. About February, '72, this eye also recovered, as he thinks, "perfectly," "as well as ever it was." About same time L. again became dim, rapidly; between tea-time and bed-time it reached its full development, and he could only see shadows. It remained as bad as this, he believes, till beginning of October, 1872 (he often tried it separately during the summer), when it began to improve, and in two weeks got a good deal better, but not so that he could read on admission. This improvement was about coincident with the second and very rapid failure of R., which in about an hour (between dinner and tea) became as bad as ever.

Never any pain in eyes.

November 14. R. very dense vitreous opacities; fundus cannot be seen in detail; reflex very uncertain.

L. Considerable opacities floating but not much obscuring choroidal reflex. Details of fundus much obscured. Disc and retinal vessels look normal. No retinal blood spots seen anywhere.

History.—At æt. 7 had whooping cough, and bled from mouth, nose, and ears, a good deal: “enough to run down from the ears.” When he had diphtheria, at æt. 9, he once vomited a moderate clot of blood. From æt. 6—7 till about 15, was very subject to bleeding from nose without cause. Has often fainted away from it. It stopped at about 15. At about 18 he often spat up blood in the morning, a little in phlegm “from the throat;” nothing of importance. Within last 12 months he began for first time to brush his teeth; was obliged to leave it off in about a month from bleeding from the gums; they bled “as soon as I touched them with the brush,” and would go on for three-quarters of an hour. His flesh very easily bruises. Has never bled seriously from a cut finger, &c. Resembles his father in complexion. His father, when a boy, used to bleed much from the nose, “so that they had difficulty to stop it.”

He has one aunt on father's side and one uncle; neither is liable to bleeding, but a daughter of the aunt (patient's paternal first cousin) was very subject to bad nose-bleeding till about æt. 23, when she married. Was much reduced by bleeding. Since marriage she has not bled. She has had no children. One sister of patient (second in family) was subject to bleeding at nose till she married, but not much since; her bleedings were considerable, but nothing like patient's. She has seven children, none of whom are known to bleed. He does not think she has ever had bad bleeding after confinements. The other sisters and brothers have not been subject to bleeding except now and then a little, “like other people.” The patient and his bleeding sister are the fairest in complexion of any, and in this respect resemble the father; patient also is said to take after father in features. Patient youngest but two of ten; 8 f. + 2 m. Has been in habit of going with women pretty regularly since about 18; at one time (early on) thinks he weakened himself by excessive indulgence, but not lately. Apparently no connection between the bleeding and the sexual indulgence (rather the reverse).

Once, however (last summer), the day afterwards, his R. eye became somewhat dim for a few days, and he fancied it was caused by the indulgence. (This attack was not mentioned above.) Scarcely ever has masturbated. Never venereal disease. Urine generally thick.

November 14. Urine, much lithates. Reaction just acid. S. g. 1042. Phosphates on boiling. No albumen.

Repeated attacks of Hæmorrhage into the Vitreous of one Eye in a Man of middle age. No special cause assigned.

The following notes were written in April, 1878. I cannot give any further particulars of the case.

A man, aged 40, a butcher (Simpson), is under my observation on account of hæmorrhage into the vitreous. He was in excellent health, when in chopping bones he found that he did not see distinctly, and could not aim; he nearly cut his fingers. He then found that one eye (the left) was nearly blind. He had not had the slightest pain. He came to me at Moorfields nine days after this occurrence (May 7, 1877). He could not count fingers, and the fundus was obscured by very large opacities in the vitreous, the remains of blood. His pupil acted well. He recovered from this attack, and could see to read again, but in February, 1878, another hæmorrhage occurred, and he became so blind that he had only perception of light. In March he had a third attack, and at present, April 17, he reads only 3 Snellen. I cannot make out any special cause. He has not had syphilis. He once suffered from piles, but at present appears to be in excellent health. He has no headache, and never had any other form of hæmorrhage. His sight varies much from day to day.

A Case of Intra-ocular Hæmorrhages into one Eye. Detachment of Retina.

J. C. J., æt. 25, student of chemistry, single, born in Lancashire. Admitted March 23, 1879, R. L. O. H., under care of Mr. Tweedy. Tall, stout, rather anæmic. General health has been good.

About eight days ago, while exerting himself, he noticed dimness come over right eye. He had been leading a sedentary life for some time before, but this day had walked and run for four or five hours. He is subject to bleeding from nose after any extra exertion. About five years ago he had this frequently. No family history of this. Mother died two years ago; she was hemiplegic. One brother died of "consumption."

R. E.—Pupil dilated under atropine. Numerous floating webs in vitreous. *Fundus*.—Large detachment of retina with well-defined upper edge (partially discoloured and opaque) in outer and lower part of fundus.

March 26. R. V. = J. 10 at 6".

L. V. = J. 1 (6"—8").

Ordered rest in bed, and—

R Liq. Hydr. Perchlor., ʒi. } t.d.
Mist. Potass. Iodid., ad ʒi. }
Empl. Lytt. to R. temple.

March 7. R.—Words of J. 6 at 8".

12. Allowed to get up, but advised to keep as quiet as possible.

15. R. V. $\frac{20}{0}$. Words of J. 4. Pupil active.

21. Small ulcer of conjunctiva of upper lid near outer canthus.

23. R. V. $\frac{6}{200}$. Words of J. 2.

26. R. pupil well dilated under atropine. V. $\frac{20}{200}$ barely, not improved by spherical glasses.

28. Stop mercurial. Mist. Potass. Iodid., ʒi, t.d.

30. R. V. $\frac{20}{100}$ and $\frac{20}{70}$ nearly. Pupil dilated. *Ophth.*

Exam.—Still considerable detachment at outer periphery, and large dark patches in vitreous, but site and outline of disc more visible; the large vessels running upwards and downwards over the disc can be made out.

April 2. Ordered a slight mercurial again.

21. E. Lyttæ to R. temple.

May 3. Yesterday and the day before he had slight epistaxis in evening—checked by cold application. R. V. $\frac{20}{70}$ and $\frac{20}{50}$ partly. J. 1 slowly at 6".

July 4. E. L. to R. temple.

15. R. V. = $\frac{20}{30}$ correctly. J. 1 well at 9".

26. R. V. $\frac{20}{20}$ almost perfectly. J. 1 well (R. = 13"). The field has improved except inwards. There is a large floating web well formed in vitreous. The detachment is much less—now almost linear down and out. Discharged May 27, 1879.

Hæmorrhage into the Anterior Chamber and Vitreous of one Eye without apparent cause. This Eye probably Myopic, the other Emmetropic.

(G. 362.) Robert K., æt. 34, came to Moorfields on April 22, 1875, with some blood in the anterior chamber of the right eye, and the history that three days before (on 19th) his wife had noticed the eye to be bloodshot, and the sight had begun to fail. It had got worse each day. When I saw him on 22nd, there was general congestion of the globe, and the anterior chamber was about one-third full of blood. The blood had sunk to the lowest part, and its upper boundary was horizontal; its lowest part was nearly black; above it gradually shaded off into the aqueous, which was everywhere more or less turbid and greenish; thus the blood was probably in great part fluid. After the use of atropine the pupil dilated partially, but there was no positive sign of iritis. The deeper parts of the eye could not be seen. There had been no pain worth mentioning. Ten-minim doses of tincture of aconite were ordered. On 26th "less blood, less congestion." 29th.—"No red blood, only slight staining of aqueous." May 6th.—Only the slightest discolouration of aqueous remained, but (after atropine) light red and white opacities could be seen by oblique illumination in the front part of the vitreous, and the fundus could not be lighted in the least; V. only shadows. During the next three months the eye did not improve; the vitreous remained full of

floating films, and the pupil covered by a thin whitish membrane; the details of the fundus could not be satisfactorily seen.

I could find no satisfactory clue to the cause of the bleeding. He believed that this eye had for two or three years been somewhat defective, for he could with it see small objects only when held close (? myopia). There was no myopia in the other eye, and with it he saw $\frac{20}{20}$. There was no history of injury nor of previous inflammation, and he was not subject to bleeding elsewhere. If it be a case of bleeding in a degenerating myopic eye, the case is still quite unusual in the occurrence of hæmorrhage into the anterior chamber.

Abundant Hæmorrhage into the Vitreous of one Eye, possibly vicarious of Menstruation. Both pupils motionless.

(G. 202.) Rachel R., a married woman of 37, was under care for nine months in 1872-73 for failure of the left eye, consequent on profuse hæmorrhage into the vitreous; the opacity produced by the bleeding was so great that no reflex whatever could be obtained from the fundus; by oblique light white and reddish flakes could be seen in the vitreous many months after the symptoms began.

The history was that the eye had begun to fail about a fortnight before admission, and that it continued to get worse until shortly after I first saw her. For a short time before and after the dimness began she had severe pain in the left temple and forehead, and sometimes in the left side of the face; there was no pain in the eyeball or side of nose. She had had no toothache. She stated that her last menstruation, which, contrary to her custom, was very profuse, had occurred four months before the eye failed. With this exception, there was no clue to the cause of her eye disease. She denied ever having been subject to epistaxis, and there was no history of bleeding from either the lungs or stomach.

The sight of the right eye was good throughout, although not quite perfect; that of the left improved only slightly while under care, on admission it was only equal to distinguishing shadows, when discharged she could count fingers at 12 inches.

The pupil of the defective eye was motionless when she first came; at the last visit, nine months later, it is noted that both pupils were rather larger than usual, slightly oval, and quite motionless even when tested in a dark room with and without a bright gas light.

This last case is an exception to the statement that hæmorrhages into the vitreous occur almost exclusively in males. It will be observed, however, that it is not an example of recurring hæmorrhages, and that only one eye was affected. It is not by any means a parallel to the cases recorded in the first part of my paper. Nor, although we may assert that as a rule menstruation saves women from the risk of hæmorrhages elsewhere, can we expect to meet with no exceptions. In this instance it will be seen that menstruation had (after a very profuse flow) been arrested for three months. I find a very important case of acute and recurring retinal hæmorrhages in a girl recorded by Mr. Power. In this instance also, menstruation had for some time been absent. Only one eye was affected, and the vitreous was not involved. Liebreich has in his Atlas published an almost parallel case. I may repeat, however, that I do not know of any case similar to that of Colin P. which has occurred in a woman.

ON THE PROGNOSIS IN CHRONIC GLAUCOMA.

(Three Clinical Lectures delivered at the Hospital in June, 1888.)

By E. NETTLESHIP.

(Continued from page 100.)

LECTURE 3.

IF chronic glaucoma have been allowed to go untreated to blindness the eye will generally remain quiet. But now and then, after a longer or shorter time, the eye becomes gradually congested and more and more painful (Case 1). The question then arises whether to try palliative measures, leeching, &c., or a conservative operation (iridectomy, sclerotomy, or optico-ciliary neurotomy), or to excise the globe. If the symptoms do not soon yield to palliatives we shall do best to enucleate if the blindness be of long standing; but if the eye have been blind only a few months a large iridectomy (or a sclerotomy) may be tried; though the symptoms will sometimes be aggravated for a time, they will often subside completely if we have patience.

There is very rarely any difficulty in deciding between a case of chronic absolute glaucoma in which the eye has become congested and painful and the secondary glaucoma set up by a tumour in the eyeball. In the earliest stages of tumour the question of glaucoma hardly ever occurs; and later, when the tumour has destroyed the sight, the eye seldom remains perfectly quiet for very long, as eyes blinded by chronic primary glaucoma so often do. Glaucoma complicating tumour has to be distinguished rather from the more rapid than from the quiet forms of the primary disease; this distinction is usually, as already stated, easy to make, but in rare cases much difficulty may arise.

I will conclude with some remarks on other, not necessarily connected, points.

First, of what is called "Hæmorrhagic Glaucoma." It is a matter of common knowledge that the prognosis is bad if retinal hæmorrhages be present before operation in a case of glaucoma. Again, if after an unsuccessful operation for glaucoma we excise the eye and find hæmorrhage, we often speak of the case as hæmorrhagic, and are satisfied. And the same term is sometimes used when the development of glaucoma has been watched in an eye previously known to have retinal hæmorrhages, hæmorrhagic retinitis, or even renal retinitis,* or embolic or thrombotic plugging of the retinal artery. Indeed, we owe several specimens showing the plug or changes consecutive to it, in the central retinal artery, to an attack of painful glaucoma coming on some weeks or months after the symptoms of embolism.† It is not likely that the relation of retinal hæmorrhage to increased tension is the same in all these sub-groups, except in so far as the onset of the congestion which may excite glaucoma is favoured by a diseased and inelastic state of the intra-ocular blood-vessels. Sudden copious bleeding into the vitreous appears sometimes to determine the onset of glaucomatous symptoms. The onset of intra-ocular hæmorrhage some time after iridectomy, as in the following case, is to be looked upon as accidental; and such a case cannot properly be called hæmorrhagic glaucoma.

CASE 45.—Mrs. Chapman, about 65 (T. I. P., 1878, p. 139). Chronic varying glaucoma of both with attacks of redness and pain; L. about three years, R. some months. L. has only p. l., T. ?+, o. d. atrophic and ? cupped.

R. $\frac{6}{12}$, F. seems full, o. d. as R., but less pale.

* For a case of glaucoma in complicated renal retinitis, see Gowers's "Medical Ophthalmoscopy," 2nd Ed., p. 326 (Case 42).

† Delafeld; Amer. Jour. Med. Sci., April, 1874. Spencer Watson; Ophthalmic Hosp. Reports, vol. viii, p. 249. Nettleship; *Ibid.*, p. 9.

Double iridectomy. Four months later sudden failure of R. from two immense sausage-shaped hæmorrhages near y. s. Later other similar ones occurred at periphery, and small ones in the other (L.) eye.

In respect to the sub-groups just indicated (of which examples are given below), it may be remarked that when glaucoma follows hæmorrhage into the retina or vitreous, or retinitis, the eye was in all probability already disposed to glaucoma; whilst when hæmorrhage accompanies or follows glaucoma it indicates diseased vessels ruptured by passive distension. Operation may easily in either case do harm by leading to fresh extravasations at the moment when the tension is reduced. Further, the congestion and oedema which follow iridectomy for glaucoma are likely to be greater and to last longer when the intra-ocular vessels are much diseased; and on this ground we can understand how in such eyes not only retinal and neural degeneration may be increased, but the glaucomatous state itself be rather aggravated than relieved by operation. A case is given further on (Case 52) showing extreme alterations in the calibre of the retinal vessels with acute oedema, both these changes having followed immediately on rapid reduction of tension from +2 to -2, by eserine.

CASES OF "HÆMORRHAGIC GLAUCOMA."

(a.) *Retinal Hæmorrhages accompanying or following the Glaucoma.*

CASE 46.—Amelia W., 58 (M., I.P., 1883, No. 91). In June, 1883, both eyes far advanced in chronic glaucoma of several months' standing. L., which had failed longest, had only p. l., T. + 1, a deep cup, and numerous retinal hæmorrhages. R. counted fingers, T. n., deep cup, F. contracted to from 5° to 10° from centre, no hæmorrhages. In view of the extravasations in R., I preferred sclerotomy to iridectomy, and performed it on both eyes on June 6.

R. did well, and six weeks later could see 19 J.

In L. iris prolapsed, and when its removal was attempted vitreous escaped; a month later, pain and + T. having returned, the eye was excised.

On opening the globe the hæmorrhages were found to be limited to the lower half of the retina, where the vessels were extremely small and diseased. The escape of vitreous at the sclerotomy pointed to a weak suspensory ligament, and the failure of the operation may perhaps be attributed rather to this complication than to the retinal disease.*

CASE 47.—John M., 69 (T., I.P., 1884, No. 312). Good health, no gout, surface arteries not hard. Advanced chronic glaucoma of at least six months' standing in both eyes, numerous retinal hæmorrhages, dilated anterior ciliary arteries, deep cups, ps. sluggish but acting well to eserine, a. cs. shallow. T. +1. $V. = \frac{3}{60}$, fs. contracted to 10° from centre. Changes all rather more in R. February, 1884, double upward sclerotomy; all went well. In two weeks T. still +1 in R., but n. in L. Not seen again, but wrote in December, 1887, that he had had to have one eye out, but could still see a very little with the other.

Probably in both these cases the hæmorrhages were a consequence of venous engorgement caused by increased tension in eyes with unsound veins. The result of the sclerotomy in the second case must be taken as relatively good. William B., 64 (T. 1879, p. 173), is a similar case in which iridectomy was done with, at any rate, no harmful result.

(b.) *Hæmorrhage found, though not previously diagnosed, in an Eye excised after unsuccessful Iridectomy or Sclerotomy.*

CASE 48.—Ellen H. (T., I.P., 1878). Here an eye, blind for two years from glaucoma, became painful, and underwent first iridectomy with very free bleeding into a. c., followed in three months by an opposite sclerotomy and a fortnight later by excision. Many hæmorrhages were found in retina between

* This case is published, with a coloured plate of the excised eye showing the localisation of the hæmorrhages, in Trans. of Ophth. Soc., vol. iv, p. 108.

equator and o. d. It is to be noted that the iridectomy hyphæma had undergone but little absorption in three months.

(c.) *Glaucoma Developing in an Eye previously known to have Retinal Hæmorrhages.*

CASE 49.—John J., 52 (T., I.P., 1886, No. 59). In March, 1885, he had hæmorrhagic retinitis in R., and just a year later the eye was glaucomatous with T. +2, and was excised for pain. In L. there was neither hæmorrhage nor glaucoma; V. $\frac{6}{6}$.

CASE 50.—Samuel B., 71 (M., I.P., 1883, No. 1916), had complained that R. had been failing for three weeks; the veins were tortuous and there were many hæmorrhages; T. ? + V. 20 J. 11 days later, November 28, 1883, T. +2, V. shadows only, subacute glaucoma. L., T. ? +, partial cataract, fundus invisible, fingers at 6". Shortly after, December 8, R. sclerotomy, L. iridectomy. December 29, R. T. +2, but no pain since operation, L. T. ? +. June, 1886, R. no p. l. T. n, L. had p. l. T. n. It is probable that the hæmorrhages in this case preceded the glaucoma.

The next Case (51) shows that hæmorrhage without glaucoma may occur in one eye and glaucoma without hæmorrhage in the other eye of the same patient and nearly at the same time. Case 7 may also be referred to.

CASE 51.—Walter T., 45 (P. 13, 209), October, 1886. L. quiet chronic glaucoma of at least a year's duration; V. $\frac{6}{18}$, F. contracted up, up-out and in, at one place lost up to 10° from fixation point. T. rather +, o. d. pale, moderately cupped all over, no hæmorrhages, veins n. R. failing for a few weeks, V. $\frac{6}{36}$, examination not completed. Iridectomy on L. Two weeks later L. had large opacities in vitreous (probably hæmorrhagic) and engorged veins but no retinal hæmorrhages. R. now examined fully and found to have severe hæmorrhagic retinitis, V. $\frac{6}{24}$ partly, T. n. No albumen or sugar. 6 to 8

weeks after operation, L. $\frac{6}{18}$, corrected by glasses; R. $\frac{6}{24}$; hæmorrhages much fewer and smaller, but veins still very tortuous.

I should add that I have not seen much of the hæmorrhages immediately following operation in ordinary acute glaucoma said by Von Graefe to be so common. But perhaps I have not looked for them often enough.

Glaucoma occurring Early in Life.

Of this we may distinguish buphthalmos, or glaucoma of infants; glaucoma secondary to keratitis, or strictly sclero-keratitis (usually in inherited syphilis); and primary glaucoma in young adults, or even in children.

I do not propose to dwell now upon the two first varieties. In the third sub-group I have included all cases of apparently primary glaucoma in which the disease began before 30, and of these I have notes of about 15. In nearly two-thirds of these cases the glaucomatous eyes were myopic. The disease began between 20 and 30, so far as could be ascertained, in 11 of the number; below 20 in only 4. Both eyes were affected in 7, one only in 6, doubtful in 1. Of the 4 below 20, the disease affected both eyes in 1, one only in 3, and the double case was quite peculiar (Case 52). Only 5 of the series of 15 were seen early enough for treatment to be useful; and all of these underwent iridectomy with good results, notwithstanding that in two of them there was considerable myopia. In none of the very young cases was there a known family history of glaucoma, but the histories in this respect are incomplete.

CASE 52.—E. Margaret T., aged 10 (P. 8. 136), was sent to me by Dr. Evan Jones, from Aberdare, in September, 1883, on account of her L. eye, which was absolutely blind with T. +2, general thinning of the ciliary region and enlargement of its trunk arteries and veins, very great dilatation of pupil

(9 mm.), large dark blood clots in the vitreous close to the retina, and also very large retinal hæmorrhages. Retina probably oedematous. The blood both in vitreous and retina most abundant in the lower half, where some of it was converted into a grey substance, o. d. deeply cupped. Retinal arteries and veins, where visible, were very small; in the upper half of the fundus the arteries were almost obliterated by concentric contraction, the lumen being barely visible, and the wall of the vessel, itself diminished in diameter, converted into relatively thick lines. The history was not very definite. A few months previously the pupil of this eye was observed to be getting larger; and the child said she had known for some time that she could not see with the eye.

The other, R., eye at the above date, September, 1883, had normal V., refraction and acc., P. also normal, T. ? +, anterior ciliary arteries tortuous and too large for her age; the o. d. showed a large, deep, rather shelving cup, and very marked, sharp, spontaneous pulsation in all the arteries. The pulsation did not amount to emptying, and it extended for a long distance into the retina,—the characters of pulsation due to aortic insufficiency rather than to increase of tension. Unfortunately, I did not measure the F. At this stage I thought it not unlikely that the order of events in L. had been (1) tendency to glaucoma, (2) faulty cardiac circulation, (3) thrombosis of retinal vessels, determined by a combination of the two above conditions, (4) rupture of vessels degenerated in consequence of the blocking.

She was an undergrown child, rather pale, shy and backward; she had had slight bronchitis 18 months before, and had never been considered strong, but had had no other illness. No history of rheumatism or ague. The eyes had not been injured. A month ago she had had some bleeding from the nose. There was no family history of blindness, but it was said that her mother's male relatives, farmers, all had an inveterate habit of going to sleep when they read. Dr. Barlow was kind enough to examine the child carefully; he found the radial pulse collapsing and the heart's impulse too strong, but there was no evidence of valvular disease, and no albumen in the urine.

In view of the peculiar changes in the L. (blind eye), and

the perfect sight and absence of symptoms in R., I did not think operation on R. advisable, but directed that the child should rest altogether from use of the eyes for three months, and see me if worse.

She was brought up again in six months (March 14, 1884), because she had been noticed for the last two or three weeks (on her return home from staying with friends) to knock against things in walking, and I was astonished to find R. now in a state of advanced glaucoma:—V. $\frac{6}{24}$, T. +3 nearly, P. 8 mm., a. c. and cornea n.; much enlargement of anterior ciliary veins, and less so of arteries, in upper part only, also slight blueness of upper part of ciliary region, but no fine congestion; o. d. deeply cupped all over, but of fair colour, arteries of normal size, pulsating as before, but pulsation now much more marked on o. d. than on retina, veins much enlarged, no hæmorrhages; F. very much contracted inwards, downwards and upwards, outwards nearly full. There had been no pain and the child had made no complaint. At this time T. of L. (blind eye) had become n. or very slightly +; a large yellowish vascular membrane was to be seen at inner half of fundus; outer half dark.

Observations on the Action of Eserine upon R. Eye.—At the visit at mid-day on Friday, March 14, I put eserine (gr. ii to 3 i) into R. eye twice in five minutes; in half an hour P. had contracted to 2.5 mm., T. was somewhat lessened and the child could read 1 J. (I had not tried hand reading before the eserine, but she said “the drops had cleared the reading.”) During the next 27 hours the father used the same eserine twice.

When I saw her at 3 P.M. on Saturday the 15th, the eye had changed in the most astonishing manner:—T. was now considerably less than in L., indeed I thought it -2; general pink congestion, P. 5.5 mm., iris discoloured (congested), V. much worse, spells 16 J., not 14 J., and cannot see $\frac{6}{60}$; the whole retina slightly hazy and œdematous; even the nerve substance of the circumference of o. d. being broader and redder than yesterday, thus lessening the diameter of the cup, which would now pass for a very large normal one; extreme dilatation and tortuosity of retinal arteries which were very nearly as large as

the veins; the veins not much larger than yesterday, but appeared lighter in colour, so that in both methods of examination there was difficulty in distinguishing some of the largest arteries from the largest veins; a small hæmorrhage at inner side of o. d.; no trace of pulsation in retinal vessels. Ordered to blister the temple, keep quiet, and omit eserine.

16th (Sunday) 3 P.M. She both felt and looked faint this morning on getting up; is liable to such attacks. R. eye; P. now 8 mm. Eye white, iris natural colour, T. considerably more than yesterday, being about = to L. (*i.e.*, n. or ?+); retina clear; disc quite altered, and is now exactly as on 14th. Retinal arteries also quite different from yesterday; in fact, about normal. No pulsation; veins still too large; several small, roundish hæmorrhages.

17th (Monday). Taken into St. Thomas's Hospital. Eserine, at first gr. $\frac{1}{2}$, subsequently gr. $\frac{1}{4}$, to $\frac{3}{4}$ l, cautiously used several times a day until the 21st. V. varied during these days, but on the whole kept decidedly better; T., however, remained about +1, and P. dilated quickly between the applications of eserine; and I therefore thought it better on the 21st to perform upward iridectomy. At this time the heart's impulse was too strong and diffused, but the sounds normal; urine free from albumen.

Though the child was not in the least dusky under the anæsthetic, the iris became suddenly discoloured as soon as the aqueous flowed off, indicating distension of its vessels.

27th. P. very wide; counts fingers at 1 foot. 31st. Sees letters of 16 J. T.n. Ophthal., no hæmorrhages seen (brief examination). April 7th. Sees letters of 6 J.; some hæmorrhages at y. s. T.n.—14th. V. $\frac{6}{36}$ partly, and words of 1 J.,

with lens and slit. F. carefully measured, and found to be much as on March 14th. After this date the eye continued to do well.

In July, T. quite n., p. still very large, a.c. too shallow; ophthal., o. d. pale all over, and cupped up to its edge, but not deeply so; artery passing down and out, represented by a shrunken white line with white branches; other arteries and veins rather too small, and coats of some of the arteries thickened. No other changes. V. $\frac{6}{18}$ partly, and spells out

1 J.; (has always been backward in reading). I have not seen her since, but have heard from her father several times, and in February, 1889 (five years after operation), her sight was reported as keeping the same, but her health not very good.

CASE 53.—Francis E., æt. 34 (T., I.P. 1884, No. 21). History of onset of acute symptoms five years ago, ceasing after a time. Fresh, probably additional, failure of V. eight months. February 1, 1884. R. eye quiet, T. +1, V. $\frac{6}{36}$, cornea, a. c., P. and F., n.; posterior polar cataract. L. eye congested; T. +3, V. $\frac{6}{60}$, Hm. 2D. $\frac{6}{24}$; P. rather larger than R., cornea steamy, a. c. n., posterior polar cataract, nasal half of F. nearly all lost. R. upward sclerotomy leaving small bridge. L. upward large iridectomy.

February, 1887. R. +1 D. sph. +1.5 D. cyl. V. $\frac{6}{18}$ F. and T. n. L. +1 D. sph. +3.5 D. cyl. V. $\frac{6}{36}$ partly; F. absent except in down-out part. T. +1; this eye has lately again had dimness, aching, and rainbows.

CASE 54.—Edmund H., æt. 33 (M., I.P. 1884, No. 408). Getting short-sighted seven years, and V. failing four years, with rainbows and pain round eyes.

March, 1884. R. T. +1?, M. 3 D. $\frac{6}{24}$; F. contracted everywhere in typical form, remaining largest outwards (inwards only 10°, outwards 50°); a. c. full depth; P. large and sluggish, and contracts badly to eserine; ophthal., deep cupping of o. d., no hæmorrhages. L. same condition, but more advanced; F. smaller; T. rather higher, and V. with -3 D. not $\frac{6}{60}$.

March 15. L. large very scleral iridectomy with Graefe's knife; severe pain for three days, when wound was gaping and full of clot. Excised ten days after iridectomy.

March 26. R. Iridectomy with keratome, smaller wound, and not so far back, but large piece of iris removed.

May. R. V. with -3.5 D. cyl. -1.25 D. sph. $\frac{6}{6}$ partly.

October, 1887. R. T. quite n. V. with $-3\cdot5$ D. cyl. $\frac{6}{12}$ eccentrically. F. much the same. Ophthal., steep cupping with marked pallor of o. d., narrow annular staphyloma.

It is worth noting that in one case of double glaucoma at about 20, with very high myopia, the patient, a male, was almost a dwarf, and obviously cretinous; and that in three others (all girls) the patients were stunted, feeble, and backward (Case 52, Ellen Bromley, Charlotte Griffin).

Glaucoma in connection with Myopia.

It is well known that glaucoma is rare in myopic eyes. When the two conditions are seen together, we find the glaucoma to have begun very early in more than half the cases (10 out of 18). Hence we must assume that in these particular instances either myopia predisposes to glaucoma, or that both are due to the same local cause; for, since youth and myopia are alike usually antagonistic to glaucoma, we should expect glaucoma to be doubly rare when these conditions co-existed; yet of the 18 cases of myopia with glaucoma only four had reached the age of 60. The lamina cribrosa of the optic disc gives way in common glaucoma because it is the most yielding part; the sclerotic in myopia yields because it is too weak; but, if a very yielding lamina cribrosa be associated with a weak sclerotic, the disc may still be the least resistant part of the capsule. In most of the cases, 10 at least, the glaucoma was double and the myopia equal in the two eyes; but there is no quantitative connection between the two conditions—as is shown by several cases. Thus, in the two cases in which the glaucoma was single, the glaucomatous eye was the more myopic in the one, and the less so in the other; in another case (Ellen Churchill, T., O.P.) the glaucomatous eye was highly myopic, the other emmetropic; whilst opposite to this we find Alice Godfrey (T., O.P.) with glaucoma in her hypermetropic R. eye, and no glaucoma in L. which has myopia of 10 D.;

again, Miss B. (P. 10, 30) has glaucoma in her R. hypermetropic eye and no glaucoma in L. which has myopia 1.5 D.

I believe the opinion is often held that when glaucoma coincides with myopia the prognosis is bad, especially in young people. That it is not so always is well shown by Cases 53 and 54 given above; but I have not had experience enough to speak at all strongly on the point. In collecting evidence on the prognosis in glaucoma with myopia, cases preceded by interstitial keratitis or "serous" iritis should be excluded.

Glaucoma following Extraction of Cataract.

I do not know much about these cases, but I believe they are generally looked upon as unfavourable. No doubt, as Mr. Treacher Collins has lately pointed out,* glaucoma, after removal of cataract, may follow exclusion of pupil by iritis, or may accompany serous iridocyclitis with deep a. c., in the ordinary way; and in the latter form the glaucomatous state is certainly sometimes only temporary, as in the case of Mrs. K. (P. 10, 153). But glaucoma may follow removal of cataract without either of these complications—perhaps as the result of attachment of the iris and capsule to the wound. It would seem also that the disease may occasionally come on so long after the cataract operation as to negative any connection between the two. Thus :—

CASE 55.—Richard P., æt. 64 (T., O.P.) had his left cataract extracted by the old flap operation, without iridectomy, when he was about 45; he stated that he could see fairly well with the eye till about 62, when it began to fail. I saw him when he was 64. T. was then +1, a. c. deep, cornea dim, V. fingers at 3 feet, P. clear and black, acting a little to light; F. absent in and down; o.d. gray and uniformly cupped. The other eye had been operated on for cataract at about the time when L. began to fail, and had done badly, P. being densely blocked. The

* O. H. R., vol. xii, p. 39.

patient was lost sight of before long, and nothing was done. I have recorded another case showing the same points in these Reports, vol. xi, p. 394 (Case 72).

Detached Observations.

Iridectomy or sclerotomy may do (or appear to do) harm in glaucoma:—

(a.) If followed by a rapid further contraction of the field, when this is already lost nearly up to fixation point (as already stated, p. 72). In congestive and hæmorrhagic cases this result might be accounted for, as already suggested, by oedema or increase of congestion, set up by the operation and acting on tissues already degenerated. (b.) When followed by hæmorrhages at the yellow-spot or into the vitreous.

But when the V. simply continues to fall, or after being stationary for a time again deteriorates, we can say no more than that the operation has failed to stop the glaucoma. This seems to be the explanation of the following case (56), in which similar failure has begun in the second eye since the operation on the first, and the first (operated) eye is worse than at the date of operation. Notice should be taken of the peculiar character of the defect of field in this case.

CASE 56.—Mr. G., 50 (P. 16, 8), complained of recurrent “inflammation” of the eyes from much reading of Arabic, which he had been learning. On February 28, 1888, he saw $\frac{6}{6}$ with each eye with -0.5 D.S., -0.5 D. cyl. I did not suspect glaucoma until I saw the discs; both were pale on the outer half, and the lower-outer quadrant of each was decidedly cupped with an overhanging edge; lamina cribrosa exposed on the cupped part; P. and a. c. n. in each. In L. the disc changes were rather more marked, and T. was slightly higher, than in R., and F. showed a considerable sectional gap at upper-inner quadrant (Fig. 10). F., R. quite full.

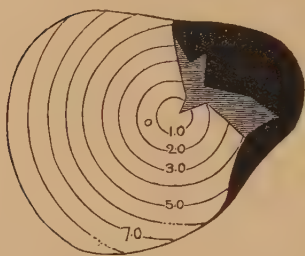


FIG. 10 (Case 56).

A week later a very large iridectomy was performed on L. In three weeks V. was $\frac{6}{6}$, partly with -4.5 D cyl. $+0.5$ D sph.

Soon afterwards he returned to his duties in a distant country, taking eserine to be used if any symptoms occurred in either eye, and with instructions to be as careful as possible. T. was then rather less in the operated (L.) eye than in the other. In July he noticed a "brown disc" above centre of F. in R., and in August discovered a new and decided gap in F. of L. near centre. The "brown disc" in R. soon developed into one or more decided gaps, which he measured roughly on an extemporised perimeter.

He now returned to England, seeking advice in the United States on his way, where several different opinions were given and fs. carefully mapped.

I saw him again in November. L. had T. n., V. $\frac{6}{6}$ with -3 D cyl., $+0.5$ D sph.; o. d. as before, but F. showed a semi-circular loss attached to the former gap and many small insular scotomata, some at about 40° , others at about 20° , from centre, and varying in density; in fact a marked tendency to ring scotoma (Fig. 11). R., T. $+\frac{1}{2}$, V. $\frac{6}{6}$, with same correction as when first seen; o. d. as before; F. shows several well-marked insular scotomata, chiefly in the upper-nasal quadrant, and all situated between 10° and 30° from centre; there was also some peripheral loss at corresponding part (Fig. 12). From the ophthalmoscopical appearances and the tension I have no doubt that the disease here is glaucoma; but my opinion on this

point is at variance with the views of some of the American surgeons who had seen the patient, and with that of my colleague, Mr. Couper, to whom I sent Mr. G. It should be added that there were no signs or symptoms of disease of the nervous system. Though I regarded the disease as glaucoma, I did not advise operation on the second (R.) eye, in view of the further failure that took place some months after operation on the operated (L.) eye.

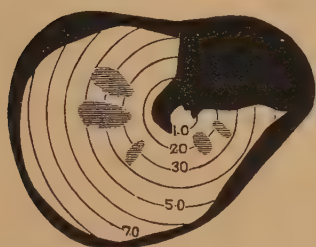


FIG. 11.

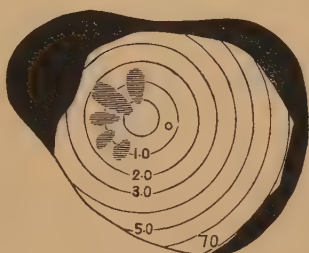


FIG. 12.

(Case 56.)

What is the pathogenetic equivalent of progressive failure after operation when T. remains (perceptibly) normal, as in the above case, we cannot at present say. The clinical side of these cases is as yet, I think, incomplete, especially as to any peculiarities they may present in mode of onset of the disease and mode of loss of F., in the early period, and in the behaviour of the wound after operation.

The failure itself is no doubt usually accounted for by progressive atrophy of the nerve-fibres of the o. n. rather than by over pressure on the retina; and this atrophy is itself, I suppose, due to continued formation of new connective tissue (Brailley).

We may, no doubt, assume a greater natural tendency to connective-tissue formation in some persons than in others, and probably also a greater tendency to persistent progress of an inflammatory change once started in a given tissue in some persons than in others. Illustrations

are seen in the varying influence of the causes producing scar-keloid, granular kidney, and cirrhosis of liver in different subjects; and in the different degrees in which a skin disease started locally may become generalised.

As to the state of the field in the early stage of cases of glaucoma simplex which eventually do badly after operation, we want more observations. I would only say here that we meet from time to time with cases of early glaucoma simplex in which the first thing observed is nearly central defect in the form of one or more scotomata near to or partly surrounding the centre, and without any contraction of the periphery. Of this Case 56 above and Cases 57 and 58 below are illustrations.*

CASE 57.—Rev. D. D., 52 (P. 11, 48). Myopic since boyhood; now (May, 1885) R., M. 7·5 D, L., M. 6·5 D, with 1 D of As. in each.

Complains of seeing "gaps" in lines of print and music lately, and that R. is defective. V. of R. corrected $\frac{6}{18}$, L. $\frac{6}{9}$. Finding a small opacity at posterior pole of each lens and no obvious changes at y. s. or at o. d., except moderate crescents, I supposed that the defect was attributable to the polar opacity, though I was a little uncomfortable in this conclusion. A year later he thought R. worse, but I found no further changes in lens, and no alteration of V., and still did not suspect glaucoma. In two years (March, 1887) R. had become nearly blind (fingers badly at 6"); o. d. very pale all over and cupped in moderate degree quite up to edge, no hæmorrhages; veins full and tortuous (suffers much from bronchitis), opacity of lens not increased; T. ?+ (rather higher than L.).

L. still saw $\frac{6}{9}$ with same lens as before (−6 D S., −1 D cyl.); T. ?+, but less than R.; o. d. rather pale, and two small vessels bent sharply at scleral border; veins tortuous as in R.; y. s. n.; opacity in lens not increased at all. Still complaining of gaps in lines and of difficulty in passing quickly from one line

* Bunge ("Gesichtsfeld," &c., 1884) mentions such cases.

to another, especially in music. F. now measured showed no contraction, but a small oval scotoma just above fixation point (Fig. 13); it was partly absolute, partly relative. October 1887. Thinks L. rather worse, especially when going up or down steps: four or five small vessels now bend at edge of o. d. V. still $\frac{6}{9}$; F. as before, no contraction but scotoma perhaps a little larger; T. n. R., T. still slightly +. Has been using eserine regularly since last visit.

I did not see him again, and he died of lung complications not very long afterwards.

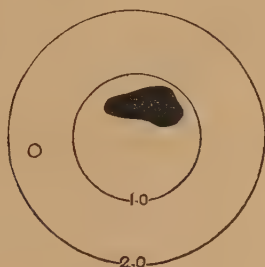


FIG. 13 (Case 57).

I do not say that these are particularly bad cases for iridectomy or sclerotomy, but the experience of the first of them (56) is suggestive. In them it is difficult to avoid the conclusion that the optic nerve is the primary seat of disease, or that at any rate it becomes involved in a progressive manner very early. These cases are amongst those as to the nature of which there is much difference of opinion, many surgeons looking upon them as peculiar varieties of primary uncomplicated atrophy of optic nerve rather than glaucoma. No doubt the point is a difficult one to settle, and perhaps we can at present only compare their features with those of ordinary cases of "primary" atrophy (progressive or not) and note the points of agreement or difference. Now, although in some of the cases of very quiet doubtful glaucoma, like Nos. 56 and 57, tension is never decidedly raised, and variations of sight

are absent, the following one (Case 58) shows that early failure of the central part of the field is compatible with both these cardinal symptoms of glaucoma, and no one could refuse to name such a case glaucoma.

CASE 58.—Wm. Barker, 48 (M., O.P.). Came first on January 28 last (1889), stating that L. had been defective from "something growing over it" for four or five years; there was a small pterygium on the cornea, and he probably attributed the defect to it. R., H. 3 D $\frac{6}{6}$; central guttate choroiditis at y.s.; T. full n.; marked physiological cupping, F. n.

L., H. 3 D $\frac{6}{60}$; T. $+\frac{1}{2}$; p. larger when shaded than R; central choroiditis as in R., not more abundant; o. d. more cupped and cupped area white and atrophic-looking; F. full, but shows a small broad central zone of absolute blindness, about 10° radius, with minute central island of imperfect vision (Fig. 14).

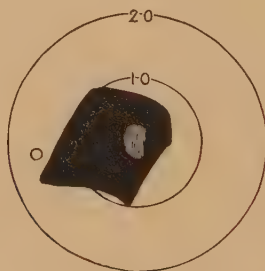


FIG. 14 (Case 58).

A month later, after continuous use of eserine to L., the scotoma seemed less dense, T. was n., and V. decidedly better (H. 2.75 D $\frac{6}{18}$ slowly). He is still under care.

Other forms of atypical loss of field are seen from time to time, but I have nothing decided to say about any such at present.

The prognosis after operation for chronic glaucoma is also influenced, as already stated (p. 98), by the patient's

state and by the behaviour of the wound; the behaviour of the wound is influenced by the condition of the patient's tissues and by the situation and size of the section. I have for some years advocated and practised a large incision, lying as much as possible in the sclerotic, and made with a Graefe's knife. Though this method is generally most satisfactory, and is theoretically correct, I do not doubt that in certain cases a wound placed further forward, and perhaps shorter, would have been followed by quicker healing, and therefore by less tendency to that form of slow inflammation which, spreading into the cornea and perhaps iris, spoils a certain number of eyes in old and feeble persons (whether the operation have been for glaucoma or for cataract). Against this must be set the risk, not so slight, of failure from removal of too small a piece of iris, or from the occurrence of anterior synechia, if the incision be made further forward and of smaller size, as may happen if a keratome be used. The point is one on which tabulated statistics would have no value; but the following two cases in each of which one eye was operated on by a colleague, whose incision was neither far back nor long, and the other eye by myself in the other manner, are, so far as they go, instructive; for in each case the eye operated by myself did badly. Case 54 in myopic eyes may also be referred to. It will be observed, however, that in the first of the two following the eye operated on by myself was in a much worse condition than its fellow; and that the second case was rather glaucoma complicating scleritis than primary glaucoma.

CASE 59.—Maria A., 69 (M., I.P., 1886, No. 934), had iridectomy on R. for subacute glaucoma with cupped disc, in May 1886; incision not far back and not very large. It did well. L. was blind from glaucoma at date of operation on R.; duration of blindness not stated, but eye said to be quiet; a month later it became painful and red with T. +3, and I now performed iridectomy, making the wound larger and further back than my colleague had made it in R. Wound remained sodden at

nasal end where a small fistulous opening remained in the sclerotic; T. for a time decidedly — . A year after operation, recurrence of pain and + T., with iritis and hypopyon; eye excised. Tissues about operation scar thickened. (? Infection through porous scar.)

CASE 60.—Jane H., 67 (M., I.P., 1886, No. 1378), underwent iridectomy on L. for “Cyclitis with + T.” of six months’ duration, in February 1886; incision not large, and not decidedly scleral; some permanent entanglement of iris; did well for about a year though o. d. was cupped and pale; then T. seems to have been rising again and F. contracting more; no later note. She came under my care in September of the same year (1886) with sclero-keratitis of upper part of other (R.) eye; T. +1 to 2, V. $\frac{6}{18}$, o. d. slightly cupped; I made a large iridectomy with Graefe’s knife, the incision far back and passing through the inflamed tissue; eye did badly from time of operation, sclero-keratitis increased, keratitis punctata appeared and V. sunk rapidly.

Whilst on the subject of the incision, let us ask what is the result when the scar is obviously a “filtering” one, *i.e.*, has to the naked eye the characters which on theoretical grounds require and justify a large and scleral incision? I assume that the deep structure of the scar allows free percolation of fluid when the conjunctiva, over and around the wound, is and remains in a state of milky or opalescent oedema, through which the lips of the scleral wound can usually be seen to be slightly separated. Sometimes a minute round fistulous opening can be easily seen somewhere in the scleral wound, and the conjunctiva over this point may be quite raised as a little vesicle. Well, some of my cases of permanently porous scar have been as favourable as possible, and others unfavourable. Of the latter, Case 59 is an instance. John N. (T. 1882, No. 367), and Maria R. (M. 1883, No. 1383), both cases of “malignant” acute glaucoma doing badly after iridectomy, had porous scars. Even in the unfavourable cases,

however, the porous scar appeared to keep the tension low, and the failure of sight was due to other and unexplained causes. I believe we may say that so much of the future as depends upon maintenance of normal tension will be distinctly favoured by a porous scar, but that, if the patient be old and his tissues degenerated, such a scar may act unfavourably by increasing the likelihood of slow inflammatory processes.*

Referring to the very inconstant relation between tension and organic damage in chronic glaucoma, we might hope to find some help in the state of the circulation: that, *e.g.*, if the heart's action were habitually feeble, or if there were aortic regurgitation, and still more if there were also marked loss of arterial elasticity, the eye tissues (retina and choroid) would suffer more from a given degree of increased tension than if the arterial blood stream were strong and well maintained. There may be much in this, but naturally it is difficult to demonstrate: Cases 52 and 7 are, however, apparently to the point. Another factor, also already spoken of, may probably be found in the natural variations of resistance in the lamina cribrosa; the more readily it yields, the greater the stretching, bending, and consequent atrophy of the nerve fibres. A question for our curators is whether such variations can be observed in sections, and whether a weak lamina cribrosa goes especially with a large deep normal excavation? As to the latter, I think it likely that a very large "physiological" cup shows a predisposition to the further yielding that will constitute glaucoma (Cases 21 and 22, 52 and 58).

Though not strictly connected with the subject of chronic glaucoma, I would just mention the influence of exposure of the eyes to cold air as one of the many causes that, by disturbing local circulation, not infrequently

* On the possibility of acute (infective) inflammation gaining access through a thin or porous scar, see a case by Mules (Oph. Soc. Trans., vol. iii, p. 58).]

decide the onset of acute glaucoma; and probably this will operate more on persons whose peripheral circulation is easily depressed or excited by other agencies than external heat and cold.

In speaking on sclerotomy (p. 100) I should have mentioned, as a peculiarity, that sometimes extreme mydriasis comes on within a few days of the operation, lasts some days, and more or less resists eserine. This occurred in the case of Bertha W. and Alfred A. (T. 1882, No. 307). No doubt extreme shrinking of iris is sometimes seen in old cases of absolute glaucoma, as in Case 61, Jane N., 39, absolute glaucoma of L. with extensive detachment of retina and extreme mydriasis: but here atrophy of iris is the cause. The mydriasis following sclerotomy must, I suppose, have a nervo-muscular origin.

I have lately (January 1889) seen almost complete disappearance of the iris follow optico-ciliary neurotomy performed on a young man, Case 61 (Edward S., T., I.P.) for the relief of pain in an eye lost by hæmorrhage (probably in part between choroid and sclerotic) after a severe blow. The pupil had been somewhat enlarged as the direct result of the blow, and had been kept still more dilated by atropine up till the date of the neurotomy. But within less than 3 weeks of the neurotomy the iris had in all parts of the circle shrunk to a scarcely visible line, and it has now remained so for several weeks; indeed it has lately become invisible. The eyeball is quite anæsthetic.

P.S.—March 15; sensibility of eye has returned to a considerable degree.

This uniform *permanent* shrinking of the iris in glaucoma comes on, so far as I know, only late and in absolute glaucoma; and perhaps (as Jane N. and Case 52 show) chiefly in eyes with detached retina or intraocular hæmorrhage in which the ciliary nerve-trunks would be stretched, or, as in the neurotomy case (61), paralysed. Is there any intrinsic neural apparatus in the ciliary muscle which, when uncontrolled by higher centres, can cause extreme mydriasis? However this may be, extreme atrophic my-

driasis in glaucoma must be distinguished from the atrophic *thinning*, with little or no *shrinking*, of patches of the iris which is sometimes seen quite early in the disease (as in Elizabeth N., 38 (T. 1883, No. 231A)), and which is one of the many evidences in favour of the belief that glaucoma of all varieties of acuteness usually starts in the ciliary region rather than in the optic nerve,

ON THE OCCURRENCE OF CENTRAL CHOROIDITIS, WITH
SLIGHT CEREBRAL SYMPTOMS, IN CHILDREN.

By EDGAR A. BROWNE.

I HAVE been struck by the fact that in a small group of cases occurring in children, a limited central choroiditis occurs, accompanied by certain general symptoms which, though capable of more than one interpretation, seem to me to be connected with cerebral disease of a definite though slight character. The common feature of this group is the occurrence of trivial cerebral symptoms in conjunction with choroidal disturbance, in the same manner as we not unfrequently see similar symptoms allied with optic neuritis. The general symptoms are slight, and easily overlooked. In the first rank may be placed night terrors. The association of nightmare in the adult with dyspeptic disturbance has led to these nocturnal terrors in children being regarded as functional, and as having a gastric origin. The prevailing characteristic of nightmare in adults is oppression, but in children it is fright. It is difficult to obtain any coherent account of these subjective sensations, but the fact that the patients have been frightened is always clear. We have a history of starting, screams, sobbing, and a terror that is often a considerable time in subsiding. A theory frequently propounded, that these attacks are due to ghost stories told by ignorant servants, generally breaks down on examination. The occurrence of vomiting still further confirms the gastric theory. But the vomiting differs rather from that which shows itself in children who are readily upset by errors in diet, as many miserable town-bred children undoubtedly are. It occurs without any obvious dietetic cause. It may sometimes be traced to over-excitement, violent games, and so forth. It occasion-

ally lasts for a period, occurring frequently perhaps during the course of three or four months, and then ceasing; to recur again at a late period, and so on. These children are often fastidious in their choice of food, and are less liable than they should be to attacks due to repletion, and so it happens that the domestic exhibition of "Gregory" has no good effects. As a matter of fact, their dietary, at all events in the wealthier classes, is most carefully superintended, yet the vomiting occurs. So with the bowels. Alternations of troublesome constipation and relaxation are noticed, and are attributed to the hot weather, fruit, or anything that comes handy. None of these symptoms are of any value taken singly, but, taken in conjunction with one another and the ocular symptoms with which they appear to me to be associated, they have a decided importance. The ocular manifestation is a choroiditis, sometimes wholly central, occasionally extending into other parts of the fundus.

It begins as a few white or buff-coloured spots in the immediate neighbourhood of or actually beneath the macula. The clots become confluent, and form small patches presenting more or less granulated or mottled surface, with some pigment disturbance. These patches may in some cases become absorbed, but in others they seem to leave small atrophic spaces with more or less well-marked central scotomata. A few spots, seldom many together, never large in area, may be seen on other parts of the fundus, never, so far as I recollect, independently of the central, though the central occurs without any peripheral disturbance.

I have always excluded the possibility of hereditary syphilis in considering these cases. The choroiditis in hereditary syphilis is more peripheral. The main stress falls on the iris, and the anterior regions of the choroid are those most frequently attacked. Exactly the reverse here. In no case have I seen any iritis. Nor in any case was there keratitis or characteristic teeth. Whether

these cases are dependent on struma or tubercle is a more difficult question. In one or two cases which I have had the best opportunity of closely observing, and have known the family history intimately, I have absolutely no clue. The parents have been without any obvious constitutional taint, and other children of the same family have not presented any marked diathesis. I have no *post-mortem* evidence, as only the children who recover have been seen by me on account of the ocular symptoms, and the links that connect these recoverable with the fatal cases (as I do not doubt there are) are at present not within my knowledge.

The connection of this kind of choroiditis with coarse though transient cerebral change is inferential. The association of double optic neuritis with meningitis is well established by numerous excellent clinical and microscopical observations, and can be verified by any one. But beyond the fatal cases, there are not a few in which well-marked cerebral symptoms and neuritis occur, and pass on to recovery. In these the association of the cerebral and ocular symptoms is commonly admitted. But it is also a matter of observation that neuritis occurs in cases in which the cerebral symptoms are very slight, but in which we certainly do not doubt there is encephalic implication. I have in my own mind distinguished a group of cases in which neuritis or atrophy occurs with the set of symptoms easily referable to gastric disturbance, and it was the observation of these cases that led me to the inference in these rarer cases of central choroidal change. It seems to me probable that certain cases of slight meningitis are transient in nature and recover, and that a proportion are never recognised at all. This can occasionally be inferred from the history of cases of early optic atrophy. Cases of this kind are those which are likely to be seen by the ophthalmic surgeon with either optic atrophy or more rarely with the central choroidal change I have attempted to describe. General practitioners probably would be

able to see this more frequently, for, as neuritis may run its course without notably affecting vision, and leaving no trace behind, so is it possible that choroidal changes may escape observation. I saw an overworked undeveloped pupil-teacher with intense asthenopia and severe headache referred in the first instance to refraction error. No relief afforded by correction. During the time she was under observation, spots of central choroiditis made their appearance, and formed a well-marked patch in each eye. Under treatment these disappeared, and the headaches were relieved. A year or two afterwards she had a recurrence of the headaches, with considerable mental disturbance, for which she went for a few months into an asylum. I saw her at intervals after with occasional headaches and asthenopia, but the choroiditis did not recur. This case, although not actually belonging to the class I am considering, yet indicates that central choroiditis may occur when we should *a priori* have expected neuritis. An illustrative case may be cited, E. H., æt. 5, a small, but fairly well-nourished, child. His sister has had ulcer of the cornea, but is chubby and well grown. He had at each macula an excavated patch, white, with faintly pigmented edges. In both eyes there are some buff-coloured fine spots, and some discrete pigment spots, of which some may be retinal. No appearance of any marked peripheral choroiditis. Between 2—3 had an attack of "irritation of the bowels," and vomited a good deal at intervals. At one time had a strange kind of fainting fit. From that time to the present has been subject to night terrors, sometimes very violent. Occasionally does not know his mother (volunteered) and seems queer, but generally a very sharp boy, active, and all functions normal. Parents have good health, are not related. Two other children healthy. This child improved in general condition under treatment, but the choroid remains unaltered.

D., æt. 11, a delicate and highly nervous girl. Both parents healthy, unrelated. One other child, very strong.

From the age of 2 subject to "bilious attacks." Bowels exceedingly troublesome, often for months constipated, then apt to be relaxed. Vomits on the slightest excitement, sometimes if fatigued. Till about the age of 8 was miserable with night terrors—used to wake up screaming and sobbing. Hysterical sobbing lasted for some time after waking. Could not give any account of her dreams or sensations. Complained of headaches, generally attributed to stomach disturbance on account of the vomiting. Has gradually grown strong, but is still nervous, and dainty about her food. Highly My.

(R. c. $-11 \text{C} - 4.170 = \frac{20}{100}$, L. c. $-11 = \frac{20}{70}$). Both eyes

ordinary myopic crescents—at the maculæ granulated central disturbance, with irregular pigmentation—over the fundus discrete circular spots of black pigment—no atrophic patches—no signs of iritis. Teeth well shaped, but brittle. This child was under observation some years, and has not altered, except gradually gaining strength. The choroiditis occurred at some period before I saw her, and has remained stationary. I think the myopia is secondary to the choroiditis, and have a suspicion in my own mind that this case illustrates the generation of a certain class of extreme myopia occurring in young people who do not employ their eyes on near objects.

B., æt. 5, a small but smart boy. Parents very fine, well-grown people, not related. When aged 3 years he was taken with vomiting, and had a comatose attack for some hours. He was very feverish. Recovered in a short time from what was considered to be a bilious attack. A few months later had a convulsion, with loss of consciousness and a good deal of twitching, though it was not noticed which side or limb were affected. Vomited at intervals, but recovered well from this attack. He had (1885) in the left eye a small central patch of buff-coloured choroiditis, another small patch some distance below the macula, and the edges of the disc very ragged and pigmented.

The central patch cleared almost away, leaving not much visible trace (V. = J. 19 held very close), but the lower patch seems permanent, and the disc has remained pale. The right eye unaffected. In this case there seems to have been some papillitis, but whether descending or spreading from the fundus I have no means of telling. I had seen this child when an infant before the attacks, and feel confident there was no choroiditis then, and no attacks have taken place since. He seems quite unaffected in intellect, and is not subject to "bilious attacks." These cases taken almost at random are seen to indicate that the central region of the choroid may if carefully watched serve to aid in the diagnosis of transient meningitis (or at least encephalic mischief). Although rare in an ophthalmic practice, I suspect they are much less common in general practice than is supposed. In all the three cases gastric disorder was definitely supposed to be the primary cause.

REMARKS ON RETRO-BULBAR NEURITIS, WITH SPECIAL
REFERENCE TO THE CONDITION OF THE LIGHT SENSE
IN THAT AFFECTION.

By G. A. BERRY, Edinburgh.

THE whole subject of retro-bulbar optic neuritis, although it has undoubtedly advanced a stage or two since Von Graefe introduced the name, is still very obscure. As has been the case in so many other instances in connection with the pathology of the nervous system, there exists with respect to retro-bulbar neuritis a dangerous tendency to lay too much stress on the data afforded by anatomical investigation. An attempt to bring the clinical facts of cases of supposed retro-bulbar neuritis into accordance with the still very imperfectly understood pathological anatomy of that affection has led de Wecker, the author of the most recent exhaustive chapter on the subject, to give an altogether fanciful description of the disease. De Wecker, without apparently entertaining the least doubt on the point, discusses cases of toxic amblyopia which are characterised by a central scotoma as coming under the heading of retro-bulbar neuritis, and forming, indeed, by far the most important group of cases recognised as due to a condition of primary inflammation of the optic nerve. Fully convinced of the existence of such an inflammation in these cases, he refers, on the one hand, to the considerable differences met with in the shape and degree of the scotoma in toxic central amblyopia, and, on the other, to the possibility of diagnosing the stage at which the inflammation has arrived in any case, and thereby obtaining valuable data for the prognosis.

From a clinical point of view, however, it is just the practical constancy in the shape of the scotoma which affords so strong an argument against inflammation in the nerve being the cause of the symptoms. This view, too, is further strengthened by the fact that the point of greatest saturation in the scotoma almost always corre-

sponds to that portion of the retina which lies midway between the optic nerve and fovea centralis. Again, a still greater difficulty on the inflammation hypothesis is to account for the practical similarity, in all undoubted cases of toxic central amblyopia, in the degree of amblyopia of the two eyes at all stages of the affection. It is difficult to imagine how an inflammation in such a situation as the optic nerve could confine itself so sharply to the impairment of the function of what is, no doubt, one particular set of fibres. More especially is this difficulty apparent when we remember that there is good reason to believe that these fibres occupy different situations in the nerve at different levels between the papilla and optic foramen. But it is still more difficult to understand how an independent inflammation in either nerve should not only involve exactly corresponding fibres, but affect their functions simultaneously to the same extent.

From these considerations, then, it can hardly be expected that any one who places the clinical facts of the case in the foreground can be prepared as yet to accept the view as to the pathology of toxic central amblyopia which has been referred to. Whether any merely circulatory disturbance, for some reason or other confined to the vessels supplying the papillo-macular fibres, and occurring symmetrically in both nerves, might account for the symptoms, can only be a matter of conjecture. Such a condition, though quite different from inflammation, might possibly give rise to anatomical changes liable to be mistaken for true inflammation.* On the other hand, anatomical investigation has definitely established the existence of such a disease as an independent destructive inflammation of the optic nerve.

Are there, then, cases whose symptoms admit of an explanation on the assumption of an inflammation of this

* The inference that inflammation has existed in the optic nerve, merely from the discovery, real or imagined, of an increase in the number of white cells or nuclei, has always appeared to me a very fallacious one.

kind? Every one is familiar with a group of cases in which, while a central scotoma exists, it is irregular in shape and subject to variation. In such cases, too, there is usually, if not always, at some time or other in the course of the affection, a distinct difference in the degree of the defect in the two eyes, or one eye alone may be affected. The affection is no more common in men than in women, or, at all events, the preponderance of cases in one sex is not marked as it is in the case of typical toxic central amblyopia. The subsequent changes met with in the optic nerve are sometimes more pronounced, too, than those ever seen in the toxic form. The whole course of such cases is then evidently more suggestive of inflammation in the nerve. Some of them are accompanied by distinct pain on moving the eye or on pressing it into the orbit, another point which is not inconsistent with the presence of inflammation somewhere in the orbit, and therefore possibly in the nerve itself. In some cases, in fact, the localisation of this pain to the nerve may be aided by the ophthalmoscopic examination. There may be changes in the papilla quite sufficiently characteristic of inflammation, and taken with the other symptoms it may fairly be inferred that the inflammation is not confined to the papilla, but exists as well over a greater or less extent of the more central portion of the optic nerve.

For cases which present either subjective or objective symptoms of such a nature as have just been described, we may, I think, fairly, so far as our present knowledge goes, diagnose *retro-bulbar neuritis*, but that diagnosis ought to be confined to such cases. As long as the inflammation only or mainly involves one particular portion of the bundle of fibres which enter into the composition of the optic nerve, some difference in the form of the amblyopic portion of the field of vision might be expected, according to its extent and level. Yet in the great majority of cases a central scotoma exists either alone, or, although it may be associated at the same time

with some peripheral defect, it is the most prominent feature. Probably, therefore, in most cases the site of the inflammation is much the same. As soon as the changes in the nerve have given rise to a central scotoma, and the objective examination is either negative or results in the discovery of some slight indistinctness of the margin of the disc, and it may be enlargement of the veins, the diagnosis may generally be made without much hesitation.

It is not improbable, considering the extent to which improvement takes place in quite pronounced cases of retro-bulbar neuritis, that a certain number of such cases may exist in such a slight degree that the defect in the centre of the field may altogether escape detection, or practically or virtually not exist at all. In this form, too, they may either persist for some time and eventually get well, or the symptoms may in the course of time become aggravated and leave little doubt as to the diagnosis. Is there any means of diagnosing these less severe forms of retro-bulbar neuritis?

It seems possible that the condition of the light sense may in some cases be of diagnostic importance. So far as my limited experience goes at present, a very decided light sense defect appears to characterise abnormal states of the macular fibres of the optic nerve, although the particular defect is probably not confined to cases of inflammation. Bjerrum was the first to point out, some years ago, the practical significance of testing the power of distinguishing differences in the intensity of illumination of two contiguous objects, as this is subject to pathological variation. He showed that the visual acuity might be diminished in an altogether abnormal manner when the difference in the luminosity of the test object and the background was lessened. Meyer has since proposed to denote this particular and distinct element in the light sense by the letters L.D., which stand for "light difference;" and this notation has been adopted by Swanzy in the second edition of his handbook on

"Diseases of the Eye." The method of testing the L.D. in the following cases, which are particularly interesting instances of defects of the light sense in this direction, was by means of the test types introduced by Bjerrum, which consist of the ordinary Snellen types printed in grey on a grey background. Bjerrum has used several different sets of types, in which the difference between the brightness of the letters and background is more or less. In the one which I have found most practical, the difference is approximately $\frac{1}{11}$; that of the ordinary letters on a white background being taken as unity.

CASE I.—In the beginning of May, 1887, a police constable, aged 42, came under my treatment at the Edinburgh Royal Infirmary, complaining of a dimness of sight in both eyes. He did not recognise people in the streets unless pretty near to them, and stated that he saw rather better in the dark. The dimness had lasted rather more than six months. On testing his vision I found that he had $\frac{20}{20}$ in both eyes, with no restriction of the fields of vision and no abnormal diminution of visual acuity in diminished light. There was no pain on moving the eyes or on pressing them back into the orbit. As the complaint of dimness of sight was so definite, I suspected that some defect must exist in his light sense. The fact that a diminution of visual acuity did not result in any abnormal manner on reducing the illumination showed that there was no defect of the nature of night-blindness, and that, therefore, although the minimum perceptible degree of illumination might possibly be somewhat greater than normal, it was not so in any marked degree. Tested with Bjerrum's (L.D. = $\frac{1}{11}$) types and with an illumination under which my own visual acuity for them remained undiminished, the patient's vision was reduced to $\frac{4}{70}$. This patient afterwards developed a well-marked central scotoma in both eyes ($V. = \frac{16}{70}$), from which he completely recovered on giving up tobacco.

CASE II.—Towards the end of last year a lady, aged 40, came to me complaining of an uncomfortable painful sensation in or behind the right eye when the eyes were moved from side to side. The pain was altogether absent when the eyes were at rest, and in consequence of this she had, more or less involuntarily, got into the way of moving her head and not her eyes when reading. She had also observed that the sensation of pain could be started by moving the head rapidly to the right, but was not felt if a similar movement were made to the left. This was in accordance with the fact elicited on examination, that adduction of the right eye caused pain, whereas abduction did not. The vision she believed to be quite unimpaired, though she had noticed that objects seen with the right eye had a dirty, not altogether sharp look, and often appeared distinctly pink in colour. On awaking at night she experienced a sensation of very bright light. This subjective sensation was of short duration, though it appeared to last “decidedly longer than a flash of lightning,” and it only occurred once after every time of waking in the dark. No subjective light sensations were noticed at any other time. The light seen was brilliant and uncoloured. The patient believed herself to be in perfect health, and knew of nothing which might account for the symptoms she had, and which she was inclined to regard as of little importance. Some years previously she had suffered for a short time from facial paralysis on the right side, which was brought on by exposure to cold. At that time there was no diplopia, nor had that symptom ever been noticed. She did not consider herself rheumatic or gouty, and had not required to have the advice of a doctor for years.

On examination I found the vision equally good in either eye, and fully $\frac{20}{20}$. Small type was read with equal facility by the two eyes, but did not look quite so black to the right eye, while at the same time the page had a distinctly pinkish colouration. There was pain on pressing the eye back into the orbit, and on moving it inwards and upwards. The ophthalmoscopic examination revealed a distinct difference between the appearance of the optic discs. The right showed a somewhat hazy surface, by which the vessels springing from it suffered slightly in the distinctness of their outline. The margin was also less

sharply defined than the left, and less so than usually characterises the normal condition. Some enlargement of the veins also undoubtedly existed. In short, the appearances were those of congestion of the papilla, although this congestion was certainly no greater than that occasionally met with when the eyes have been overstrained. It existed, however, only in the one eye, and in that in which the pain was experienced. The diagnosis, retro-bulbar neuritis, was therefore, from the description given, no doubt justified. I recommended the patient not to drive in an open carriage, as she had been in the habit of doing, but to take as much outdoor exercise walking as she cared to, to be careful as to her diet, not to read, to use dark glasses outside, and to take salicin. As she was rather opposed to counter-irritation, I did not press it. At the next visit 10 days afterwards the pain had altogether gone, but she informed me that the vision appeared to her to be less clear. On examination it was found to be as good as before, fully $\frac{20}{20}$. The coloured wool

test revealed no colour confusion at all characteristic of any form of colour blindness, but every colour appeared less saturated when seen with the right eye than with the left; blue hues mostly so, and yellow least. There was also a slight difficulty in distinguishing between certain shades of green and blue.

Bjerrum's test-types $\left(\text{L.D.} = \frac{1}{11} \right)$ were altogether invisible, even when placed at a distance of only 3 feet. At the same time there appeared to be no greater difficulty experienced in reading with the right eye by the aid of a faint illumination than with the left; there was therefore no marked night blindness.

The patient eventually recovered completely. The vision tested with Bjerrum's types was as good as my own, *i.e.*, nearly $\frac{20}{20}$, while all colours appeared equally saturated to both eyes. The appearance of the disc, too, became so nearly normal that it would now be difficult to detect any difference as compared with that of the other eye.

So marked a light sense defect of a nature similar to that exhibited by these two cases is, so far as my experience goes, far from common. It is certainly rare to find

full visual acuity tested in the ordinary way along with so great a difficulty in distinguishing between luminous impressions of different intensity. This kind of light sense defect is known to characterise those forms of amblyopia which are the result of some change in the peripheral nerve elements of the visual apparatus. On the other hand, alterations in the pigment cells of the retina, and possibly circulatory disturbances in the choroid, give rise to more or less pronounced difficulty in the perception of feebler intensities of illumination. This distinction appears first to have been clearly made by Bjerrum, and may no doubt be of diagnostic importance.

From the defect met with then in the two cases just recorded it might be assumed, even did the other symptoms not point in the same direction, that some disturbance existed in the respective optic nerves. I have at different times made examinations into the state of the light sense in various forms of amblyopia; but, although I have met with a very distinct diminution in the acuteness of the appreciation of differences of intensity of illumination, these two cases are so far the only cases I remember to have seen in which a defect in this respect existed in such a marked degree without any appreciable alteration in the form sense in ordinary daylight, and for black objects on a white background. With regard to toxic amblyopia in general, I have referred in a previous volume of these reports* to the test with Masson's disc. By means of this test I have satisfied myself that a similar defect in the light sense usually exists in that affection, and since using Bjerrum's types, I have been able to confirm this by that test as well. Yet the disproportion between the defect and the visual acuity was never found to be so great as in the first case here referred to. Perhaps this may be owing to one's rarely having an opportunity of seeing the very early stages of toxic amblyopia. So far as the condition of the

* Vol. x, part 1, 1880.

light sense goes, then, the presumption is that the lesion to which toxic central amblyopia is due, whatever it may be, is located in the nerves, and not more centrally, as there are, perhaps, other reasons for believing to be more probable.

It is interesting to note that the particular defect was still more marked in the second case, which was one of retro-bulbar optic neuritis, or, at all events, congestion. In other forms of neuritis, too, which are not characterised by any disproportionate impairment of the functions of the central portion of the retina, there exists the same defect in the L.D., but to an extent which, though no doubt varying considerably in different cases, probably never amounts to anything like that found in the two cases described.

To these two cases of light sense defect, the one occurring at the beginning of what turned out to be apparently a typical case of tobacco amblyopia, and the other constituting the most marked subjective symptom of a slight retro-bulbar neuritis, I am able to add another which was met with in a case of a somewhat obscure though no doubt allied form of amblyopia, from which there has been almost complete recovery.

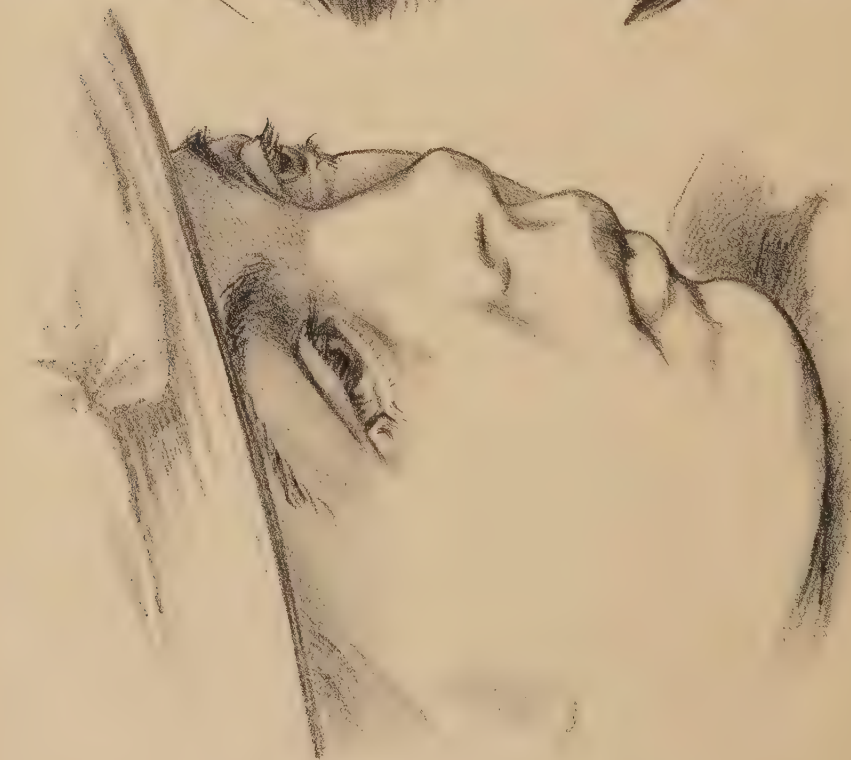
CASE III.—George C., æt. 25, a gardener, began to complain of failing sight towards the beginning of 1887. The blindness reached its full height about three weeks afterwards, and continued much the same up to the end of April, 1888, when he first came under my care at the Edinburgh Royal Infirmary. At that time his vision was about $\frac{8}{200}$ in either eye. There was no restriction of the fields of vision, and no definite central scotoma in either eye. Colour vision was very defective. The discs were markedly pale and presented altogether the appearance of post-papillitic atrophy. The patient never drank to excess, and smoked rarely more than 3 ozs. a week of "twist" tobacco. He was ordered iron and quinine, which he took for three or four weeks and then discontinued, as he did not find

that his sight improved. I next saw him in January of this year. His vision is now $\frac{18}{30}$ in either eye. The fields are not restricted, and colours appear only slightly hazy. He complains, however, of not being able to recognise people until they speak to him. On testing him with Bjerrum's types, I found his V. = $\frac{2}{100}$ (for L.D. = $\frac{1}{11}$), and $\frac{12}{200}$ (for L.D. = $\frac{1}{6}$). He has therefore a very marked light difference defect. The discs are now both extremely pale, and the vessels covered here and there by a densely white deposit. He still continues to smoke at least 2 ozs. a week of the same tobacco. He had an attack of typhoid fever in February, 1888, shortly before and after which he saw Mr. Nettleship. From the notes of the case at that time kindly supplied to me by Mr. Nettleship, I find that he could only count fingers at 18", so that when I first saw him he had begun to improve. He himself ascribes the improvement in his sight to his recovery from the typhoid fever. It seems not impossible that this recovery may have been in some way connected with the visual improvement.

The point of interest in this case in the present connection is the fact of the great defect in the light sense remaining after such a marked improvement in vision tested in the ordinary way had taken place. It explains, too, the difficulty which the patient has in recognising people, as the difference in luminosity of the different portions of the face is small compared to the contrast presented by black letters on a white background.

The cases show the importance of testing the light sense without any attempt at dissociating it from the test for visual acuity. It is evident that when the L.D. is abnormally defective it will have a great influence on the visual acuity, as tested by Bjerrum's types. Further, it is obviously only necessary for this, that the defect should exist for the central portion of the retina. We may conclude, therefore, that *a very much diminished acuity when the light difference is reduced, coexisting with otherwise normal or good visual acuity, is indicative not only of some affection of the*

optic nerve, but of one which involves mainly the fibres which supply the centre of the retina. The actual lesion which may produce this effect need not always be identical in nature. Possibly a similar condition of the peripheral light sense exists for those cases of neuritis which primarily lead to restriction of the field of vision, but as to this I have no experience; the practical testing of the peripheral light sense is, besides, by no means easy.



W A S del

Fig 1

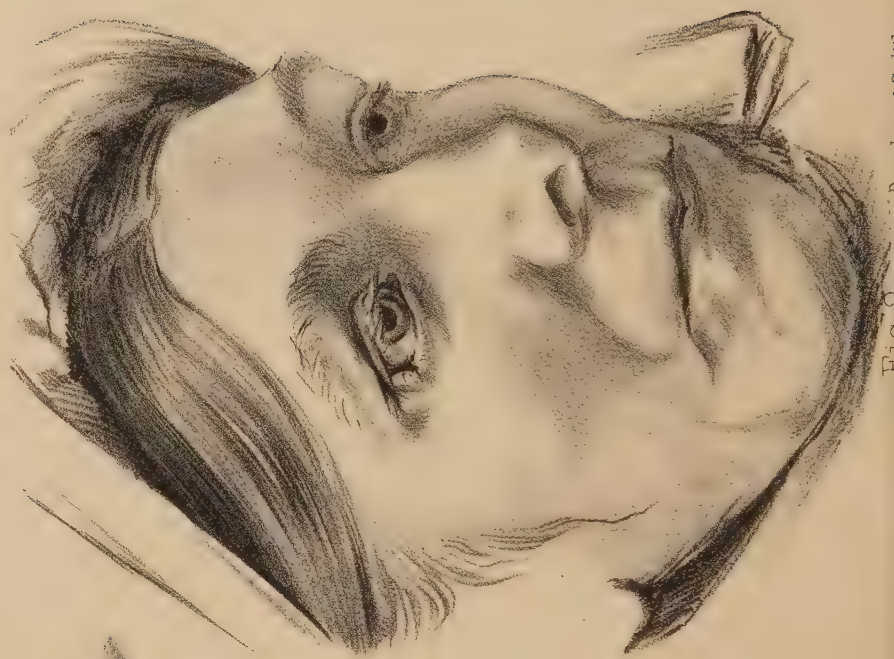


Fig 2

NOTE ON A CONGENITAL DEFECT (? COLOBOMA) OF THE
LOWER LID.

By G. A. BERRY, Edinburgh.

THE accompanying drawings exhibit a variety of congenital defect in the formation of the lower lid, which appears to be of very rare occurrence. I have been unable to find any description of a similar case in ophthalmic literature.

Fig. 1 gives a very good representation of the right lower lid of Janet R., a girl of 15, whom I first saw on account of an acute catarrhal conjunctivitis. On the margin of the lower lid, at about the junction of its outer fifth with the inner four-fifths, is a depression which has all the appearance of a badly united cut in this situation, and which is continued into a cicatrix on the skin surface, about 4 mm. in length. The natural curve of the lid is somewhat altered, the outer portion passing almost vertically downwards to the notch.

On inquiring whether she had at any time received an injury to the lid, she told me that she was born with the defect, and that her mother presented exactly the same appearance in both lower lids. This statement I was able to confirm, and Fig. 2 represents the appearance in the case of Mrs. R. The defect exists in the lower lid of both eyes *absolutely symmetrically*, and exactly similar to that in the daughter's right lower lid. Mrs. R. had, further, a hare-lip, which was operated on when she was eight years old. Of this condition there still remain a well-marked cicatrix and a notch which is represented in the drawing. She has had three children, two of them, Janet and a brother, being twins, and an elder daughter. The elder daughter I have seen; she is entirely free from either deformity. The son was born with hare-lip. Of Mrs. R.'s

brothers and sisters, only one, a brother, has any defect of the lid. The defect is in the lower lid of one eye, and, according to Mrs. R.'s description, is quite similar to her own. She does not remember a time when it did not exist, but her daughter declares that her uncle asserts it was due to an accident. There is, therefore, some doubt as to this case; and, as I have not seen him, I am unable to say whether it is a congenital deformity or not, although the possibility of such being the case is, perhaps, worth mentioning.

There are two points of interest in connection with these cases, viz., the hereditary transmission of the deformity and the difficulty there seems to be to explain its nature. The one point, too, appears to me to have some bearing on the other. The exact similarity of the condition in both parent and child, taken together with the fact that the other twin was born with hare-lip, seems almost to exclude the possibility of its being the result of any accident occurring during intra-uterine life. These same facts are very suggestive, too, of the lid defect being caused by some arrest of development or to be, in other words, a true coloboma. This view is also strengthened by its bilateral occurrence in the one patient. On the other hand, it is totally different from other cases of supposed coloboma of the lid which have been described. It is not evident either at what time in foetal life the particular arrest in development might possibly take place, as its position does not coincide with any known fissure, or, indeed, in any way with foetal anatomy.

About 50 cases of coloboma of the lid have been recorded, all of which seem to have been to the inner side. By far the greatest number have involved only the upper lid, and about one-third of all the cases have been symmetrical on the two sides. A certain proportion, too, of the recorded cases have been accompanied by hare-lip or coloboma of the iris. Several hypotheses have been advanced to account for the so-called coloboma of the

lids; but, as these cases have probably little bearing on those here recorded, those interested in the subject may be referred to a paper by Dor, published in the Transactions of the last International Ophthalmological Congress, where the different views are clearly discussed.

AMAUROSIS (WITHOUT OPTIC NEURITIS) DUE TO
CEREBRAL TUMOUR.

By J. HUTCHINSON, JUNR.,

Clinical Assistant at the Hospital.

THE following case presents several points of special interest which justify its record in detail, especially since the eye symptoms were the first ones which attracted attention, and the diagnosis presented considerable difficulty.

The patient, a young labourer, aged 20, first attended the Hertford Infirmary; three weeks after this he came to Mr. W. Tay's clinic at Moorfields, and was then transferred to the London Hospital, where he was under the care of Dr. Warner. To the latter, and to the house physician, Mr. Debenham, I am indebted for various notes of the case, and for the opportunity of obtaining the optic nerves after the patient's death.

George A., a brickmaker by trade, living at Ware, in Hertfordshire, was said to have been quite healthy until the spring of 1888, when his sight began to deteriorate, and gradually became worse day by day. He noticed also some general weakness, he became more tired in walking than formerly, and after a time the eyes and head began to ache. At first intermittent, this headache became almost constant. He was admitted into the Hertford Infirmary on June 25th on account of his loss of sight, which affected both eyes, and at that date was so advanced that "he could not see to read large type, even when held close to the eyes." The "tops of the eyes ached," and on June 30th he vomited once. The vision in the right eye somewhat improved, so that on the 28th he could make out large letters; the defect in the left eye did not alter much.

I do not know that any diagnosis was made in the case until he came to Moorfields Hospital on the 11th of

July, when tobacco-amaurosis was at first suspected, for the following reasons:—

1. The failure had been gradual, and was wholly unaccounted for by anything seen in the fundi. It was thought that the discs were a little hazy, but, personally, I could see nothing abnormal in them, and this was later confirmed by other observers. Both eyes were emmetropic.

2. When tested by the perimeter a definite central scotoma for red and green was made out.

3. He stated that he was in the habit of smoking about half an ounce of shag daily.

But on investigating the case it seemed very improbable that it was really one of toxic amblyopia. He was of an age (only twenty) at which amaurosis from tobacco is extremely rare, his visual power (only 20 J. badly at 10" and $\frac{1}{60}$) was unusually impaired, and there was some evidence of mental deterioration in addition to his complaint of constant headache and loss of sleep.

It does not, perhaps, mean much in a country labourer to have a heavy and stupid expression, but besides this the patient was liable to break into causeless laughter, and answered questions in a rather slow and imperfect manner. But perhaps the most conclusive argument was the state of his pupils, which were large, and acted very slowly both to light and on accommodation.

Nevertheless, on the chance that the visual defect might be due to tobacco-amaurosis, he was ordered to abstain from smoking, and to take a mixture containing tinctures of nux vomica and perchloride of iron. He returned six weeks later, when his vision had, if anything, become rather worse, although he had entirely left off smoking.

On August 23rd I made the following note:—"Fundi normal. Sickness after taking food, occasional faintness, still much headache. Some deafness on the right side.

Knee-jerks normal, finer movements of hands perfectly executed. (?) Cerebral tumour."

There was nothing in the family history to throw light upon the case; there was no history of tumours, his father and mother were alive and healthy, and there was no evidence to point to syphilis, either acquired or inherited.

He was admitted into the London Hospital on the same day, and died suddenly on September 15th—three weeks later, and probably about four months since the first symptom developed. His temperature and urine were normal until the end, but his mental condition altered considerably. The headache became constant with exacerbations, the pain being most severe in the eyes and towards the occiput. The pupils, at first contracting to light in a sluggish manner, ultimately became fixed; his vision varied somewhat (at times he could not see the window even). Partly owing to the headache it became very difficult to examine the fundi, but when they were looked at there was not the slightest evidence of neuritis. There was only occasional vomiting, but frequent nausea. The knee-jerks continued normal or were slightly exaggerated, and the other reflexes (triceps, plantar, &c.) were increased.

Treatment consisted in the administration of mercury and iodide of potassium, the application of ice to the head, and various sedatives were tried, without effect, with the object of relieving the headache.

The *post-mortem* examination was as follows:—

Viscera healthy, with the exception of the brain. Heart in systole. A large soft glioma involved the apex of the right temporo-sphenoidal lobe, and reached across the middle line in the inter-peduncular space. "On trying to examine the brain more closely it was found to be too soft to allow of the exact limits of the tumour to be made out, but the growth had penetrated somewhat deeply into the right crus cerebri; at the base of the brain it extended

over an area as large as the palm of the hand, and about an inch and a half in depth" (Dr. F. J. Smith).

The optic nerves and posterior halves of the globes were removed, and I made sections involving the optic papillæ as well as transverse sections of the optic nerves, both near to the eyes and towards the chiasma.

The symptoms had led me to suspect that, though the ocular expansion of the nerve might not be inflamed, we should find retro-bulbar neuritis, and possibly that the inflammatory changes would chiefly affect the band of fibres concerned in tobacco-amaurosis. The results of examination (which Mr. E. T. Collins kindly confirmed), however, proved that the nerve in transverse sections was but slightly and equally affected throughout. The perineural sheath was filled up with small cells and delicate connective tissue, which, there was no doubt, consisted in a prolongation of the gliomatous structure downwards from the base of the brain towards the globes. Immediately behind the lamina cribrosa was also a similar infiltration, the papilla itself being quite normal. In the interstitial bands there was but little evidence of change, nor could there be said to be any true neuritis. There was no distension of the nerve-sheath, either close to the eye or further back.

Looking at the slight character of the changes present, it seems most probable that the amaurosis was due to involvement of the optic chiasma or tracts in the growth, and I am sorry that the extremely soft nature of the tumour prevented an accurate opinion being formed on this point.

The case recorded is the exact converse of those to which Dr. Hughlings Jackson has especially drawn attention—examples of optic neuritis or papillitis from cerebral tumour in which (at any rate for a time) there is little or no defect of vision. Here we have a large, soft, rapidly growing basal tumour, causing great defect of sight but no papillitis. It is of course possible that, had

the patient survived a few months more, optic neuritis would have developed. So far as one can judge from the symptoms, the tumour had started four or five months before the patient's death.

Are such cases not infrequent? According to Aunuske and Reich, papillitis is absent in only about 4 per cent. of the total number of cases of cerebral tumour. It would be of interest to know in what proportion of the 4 per cent. amaurosis was present without changes visible to the ophthalmoscope.

I do not wish to suggest that such a case has any bearing upon the theory of the causation of optic neuritis so well advocated by Messrs. Lawford and Edmunds. On the *post-mortem* table a soft infiltrating glioma at the base of the brain may appear to be closely similar to meningitis; in reality they may be wholly different, and one may be much more liable to produce optic neuritis than the other.

NOTES OF CASES OF EPITHELIOMA AND SARCOMA
AFFECTING THE CORNEA AND CONJUNCTIVA.

By J. B. LAWFORD,

Clinical Assistant at the Hospital.

THE following brief notes concern some pathological specimens which came into my hands while Curator of the Moorfields Hospital Museum.

Three of these were obtained from patients in the Hospital, and one from a patient under care elsewhere. As they were all cases of some rarity, and of both clinical and pathological interest, it seemed worth while to publish them in these Reports; I have considered them, however, almost entirely from their pathological side.

The history of Cases 1 and 4 have been obtained up to date; of Cases 2 and 3 unfortunately nothing is known since the operation.

CASE 1.—*Epithelioma growing at Corneal Margin.*

F. G., æt. 66, a patient under the care of Mr. Nettleship, who has kindly given me the notes of the case.

In May, 1887, whilst under treatment for bronchitis by his regular medical attendant, a "spot" was noticed on the "white of the right eye." Some little time afterwards the patient consulted an ophthalmic surgeon, and was advised treatment under which the eye "got well." It soon relapsed, however, and on again consulting the surgeon he was told that the spot was "probably cancerous." A year previously the head of a burning match flew into the right eye, and caused much pain for a short time.

In November, 1887, he was seen by Mr. Nettleship. The condition at that time was as follows:—"Right eye. At the outer (temporal) part is a thickened patch encroaching slightly on the cornea. It is rather buff coloured, its texture a little translucent, and its surface decidedly rough or (?) papillary. Its

greatest thickness is just at the limbus, the scleral part is movable with the conjunctiva, the corneal part is fixed. In the vicinity the anterior ciliary and conjunctival vessel trunks are much enlarged. The eye is in all other respects sound."

The growth was removed, under cocaine, and the galvano-cautery applied to the corneal margin, and to the floor of the hollow which was left. The wound healed slowly by granulation, and up to the present date (January, 1889) there has been no return of the growth, and the eye has remained quite sound.

The growth when removed consisted of a strip of greyish-looking tissue, measuring rather more than 0.5 mm. in its thickest part. Examined with the microscope, the surface of the tissue is uneven, with small papillary projections and intervening hollows. In sections (cut at right angles to the surface, *see* Fig. 1) a deeper fibrous part and a superficial cellular part are evident. The former has been very slightly coloured by the logwood; it contains few oval nuclei, and in its deeper layers is loose and irregular in arrangement. Adjoining the cellular part it is more like corneal tissue, which it probably represents. There is nothing which can be recognised as the anterior elastic lamina of the cornea, but the transition from the fibrous to the cellular tissue is abrupt. The epithelial part, which comprises rather more than half the thickness of the tissue removed, consists of cells which vary in shape and size much as do the cells of the normal corneal epithelium. The cells resting on the fibrous tissue are very similar to those of the deepest layer of the corneal epithelium, but are less regular in form and arrangement, and more closely packed; their nuclei stain deeply. Superficial to these are cells more circular in shape, less closely set, and the nuclei of which stain badly.

More anteriorly still the cells gradually become more degenerate looking, the surface layers being composed of long fibre-like cells, which are scarcely at all coloured by the staining fluid. The cells of these layers are not very closely adherent, and numerous small spaces are evident where they have become separated. These cornified cells in places dip into the deeper layers.

There is a considerable amount of stroma throughout the

growth; in the middle layers and towards the surface this is most evident, for here the cells have fallen out in many places, whereas in the deep layers they remain in position. The stroma has a faintly granular appearance, and does not stain with hæmatoxylin. Several quite characteristic "bird-nest" collections of cells can be seen in the sections (*see* Fig. 1, *n*).

The growth is apparently a-vascular. No vessels can be found in any of the sections in the cellular portion.

There seems to be but little tendency to invasion of the fibrous tissue by the epithelial elements, but here and there a small tongue dips into the subjacent tissue.

CASE 2.—*Epithelioma at Corneal Margin.*

George L., æt. 65, sailor (Reg. No. 2456a), was admitted to the Hospital under Mr. Gunn's care on May 10, 1887. For about twelve months he had noticed a small spot on his left eye, which he thought was increasing in size.

On admission, there was a pale, slightly vascular swelling at the sclero-corneal junction of the left eye, on the temporal side, to which some enlarged vessels ran from the conjunctiva. The swelling seemed to consist of two or three solid semi-translucent outgrowths. The eye was sound in other respects.

There was a small ulcerated spot on the left ala nasi, and a little nodule in the skin of the left side of the nose.

There were no enlarged glands.

Family history good.

The growth was dissected off, and after removal consisted of a narrow strip of rather hard greyish tissue; the portion removed from the cornea was somewhat thicker than the outer or episcleral portion. After hardening in Müller's fluid, vertical sections of the growth were made and stained with hæmatoxylin.

Microscopic Examination.—Beneath the epithelial portion is some loose connective tissue with numerous nuclei, at what is apparently the outermost or conjunctival part of the growth; to the inner side of this is tissue resembling healthy cornea, but containing more nuclei.

Between this and the epithelial cells is a sharply defined layer, which is doubtless the anterior elastic lamina of the

cornea. The cells in contact with this layer are not unlike those of the deepest layers of the cornea, but are not so columnar in shape; they are succeeded more superficially by circular and flattened cells, those on the surface being thin and fibrous looking. The deeper layers stain well, the superficial ones badly.

Among the anterior layers of cells are several areas where considerable degeneration has occurred; here many of the cells have disappeared, and others are shrunk and degenerate. Spaces are thus formed, which are crossed irregularly by strands of stroma. Except at these points the stroma is not very evident; but where seen it has a more fibrillated appearance than in the previous specimen, and is more coloured by the log-wood. There are several characteristic "bird's-nests." In the outer or conjunctival part there are in two or three places small invasions of the subjacent tissues by the epithelial cells; these processes or buds are continuous with the main growth, and have, apparently, no connection with the underlying tissues into which they merely push their way. The growth is apparently completely devoid of blood-vessels. In the subjacent connective tissue there are a few small blood-vessels and some extravasated blood.

CASE 3.—*Ulcerating Epithelioma of Cornea.*

Joseph E., æt. 49, was admitted to Moorfields under Mr. Tweedy's care, January 30th, 1885. His right eye had been painful and inflamed for six months. On admission (notes by Mr. E. Hudson), "the right eye was much congested; there was a spreading ulcer of cornea at nasal side covering an area from the inner edge of cornea to a point over the inner pupillary edge of iris. The border of the ulcer was infiltrated, and the ulcerated surface had a gelatinous appearance. There was no iritis; T. n. The left eye was healthy." Mr. Tweedy suspected that the disease was epitheliomatous. The ulceration continued to spread outwards across the cornea in spite of active treatment. Scraping, cauterising, the local use of iodoform, quinine, mercury, and other remedies seemed to have no effect in arresting the ulcerative process. On April 21st, after nearly three months' treatment in the hospital, the eyeball was

excised; the ulceration had then involved the whole superficies of the cornea, with the exception of a small irregular area near the upper and outer margin, which showed as a little island of moderately clear corneal tissue raised above the level of the surrounding structures.

The naked eye appearances of the eyeball after removal were as follows:—Size and contour normal. The cornea is uneven on the surface and semi-opaque. In nearly its whole extent the appearance is that of cicatricial tissue, but at the upper outer part is a small area with steep, slightly undermined edges which has escaped the ulcerative process. The thickness of the cornea varies; it is thinnest at its upper part. On the cicatrised part some large blood-vessels are visible.

No adhesions between iris and lens capsule, but on the latter in the pupillary area is a thin layer of lymph. Iris not appreciably thickened. Lens *in situ*. Cortical layers clear, nucleus slightly hazy. Vitreous clear and of normal consistence. Retina and choroid apparently healthy.

Sections of the cornea were made with a freezing microtome and stained with logwood. Microscopical examination gave the following results:—

The thickness of the cornea varies considerably; in general the true corneal tissue is reduced in depth, though the measurement of all that macroscopically represents the cornea is in places greater than that of a healthy specimen. The posterior layers of the cornea are fairly normal, exhibiting merely some increased nucleation (*see* Figs. 2 and 3), and in the peripheral part a few new vessels. The posterior epithelial layer is wanting in greater part, and has not improbably been removed during the manipulation of the specimens; however, in places where it remains adherent, the cells have a slightly swollen, indistinct look, and may have undergone some pathological change. At only one part, and over a comparatively small area, is the anterior elastic lamina (Bowman's layer) evident. This portion of the cornea forms a kind of plateau or flat-topped eminence, slightly raised above the surrounding surface, and probably corresponds to the area noticed before excision, over which ulceration had not spread. The anterior epithelium over this area is considerably altered, the several rows of cells being much less regular than in healthy eyes, and not so easily

differentiated, and the whole layer separates from the elastic lamina much too readily.

The anterior layers of the cornea, excepting over the area above described, are replaced by tissue consisting almost entirely of small, round cells which stain darkly with logwood—a sort of granulation tissue. In this layer and in the corneal laminae immediately subjacent are numerous small blood-vessels filled with corpuscles. Superficial to this granulation tissue, as it may be called, is a layer of very varying thickness composed almost entirely of well-formed nucleated epithelial cells resembling in size and appearance the cells of the middle layer of healthy corneal epithelium (*see* Figs. 2 and 3). I have said “almost entirely,” because in some places, notably on one side of the plateau spoken of above, these cells appear somewhat irregular in shape and not so well defined as elsewhere. This epithelial layer is in parts of greater thickness than the corneal tissue beneath; from it numerous columns or rod-like extensions of epithelium pass into the underlying tissue and make their way along it in different directions; some, seen in transverse section, appear as cylinders of cells, others are shown in longitudinal section. A few typical “bird-nest” clusters are visible. There are no blood-vessels to be seen in the epithelial layer, but in some places small vessels with some surrounding tissue have become enclosed by epithelium. This epithelial layer is directly continuous with the conjunctival epithelium; but except over corneal tissue it shows no tendency to invade the strata beneath; over the whole area of the cornea, however, its invading properties are very evident. Its surface cells are flat, but not so flat or horny looking as the most anterior layers of the healthy epithelium.

There seems but little doubt that this case was one of an unusual form of epithelioma of the cornea in which extensive ulceration had occurred; the tendency of this ulceration having been to spread along rather than deeply into the cornea. The disease seems to have begun at the corneal limbus, as epithelioma generally does, and from this starting point to have extended steadily across the cornea, the clinical features it presented being those of ulceration rather than of new growth. Although the

superficial layers of the cornea were destroyed, the deeper layers were scarcely affected; from the microscopic characters of the disease, however, there is no reason to doubt that extension to these layers would have occurred had the eyeball not been removed.

I have been unable to find records of any cases like the above, but I have not had an opportunity of looking up all the references I obtained. The cases of epithelioma of cornea (not very numerous), which have been published by both British and foreign writers, were cases in which there has been an evident new growth, something which could be dissected off the cornea. I have never seen a specimen like that here described, and the only one which, from its naked eye appearances, I thought might exhibit microscopically a similar condition, proved when examined to be of quite a different nature.

Of the clinical features of the case I cannot speak. I have given above a few brief notes taken from the in-patient record; that the symptoms were in some respects unlike those of ordinary serpiginous ulceration of cornea is apparent from the fact that the surgeon under whose care the patient was suspected the disease to be epitheliomatous. This case suggests that it is not altogether improbable that some of the cases of very intractable serpiginous ulcer of cornea, occurring in people beyond middle age, may in reality be cases of epithelioma.

CASE 4.—*Sarcoma at Sclero-corneal Junction. Removal. Early Recurrence.*

Edward R., æt. 38, labourer (Reg. No. 2354), was admitted to Moorfields Hospital, under Mr. Tay's care, June 13, 1887.

Ten years previously he had noticed a small red spot in his right eye. Six years ago he had this spot "burnt with blue-stone," and states that after this treatment it disappeared but returned in three years' time. It did not, however, increase much in size till Christmas 1886. An operation was then performed and the growth partly removed; since then a rapid

increase in its size has occurred. There is no family history of importance; the patient has always had good health. On admission the following notes of his condition were made by Mr. Collins, the then Senior House-Surgeon:—"Right eye. Down and out, partly on the cornea, partly on the conjunctiva, is a reddish granular mass about the size of a small bean. It does not enter the anterior chamber, nor is the cornea infiltrated at its margins. Its surface is smooth and apparently covered by an epithelial layer. $V. = \frac{6}{6}$. T. n. No ophthalmoscopic changes."

The growth, after removal, consisted of a fairly hard nodule, somewhat kidney shaped, its concave surface slightly irregular, its convex surface smooth. It measured in its longest diameter 5 mm. After hardening in Müller's fluid, the specimen was frozen and sections made with a microtome.

Microscopic Examination.—The growth is a sarcoma, consisting of cells which vary somewhat in size and are imbedded in an almost homogeneous matrix. The cells are oval or oat-shaped, but all of small size; no large spindles are evident in the sections examined. The stroma is in moderate amount, but the closeness with which the cellular elements are packed varies a little in different parts of the tumour and also, probably, according to the way in which they are seen, whether in longitudinal or cross section. An epithelial layer is discernible over nearly the whole convex surface of the nodule, and in some parts where this is wanting it is doubtful whether the growth has made its way through this layer or whether the epithelium has become detached during preparation of the specimen. The appearance of the layer is more like that of the conjunctival than of the corneal epithelium; the cells are generally long and flat but in places they look swollen. Between the epithelium and the sarcoma cells is a layer of varying thickness containing large numbers of small round cells, considerably smaller than those of the tumour, from which they also differ in shape. In this layer towards one end of the growth (upper or lower) is some blood extravasation, and at the base of the growth, in some loose connective tissue attached to it, there is a large amount of extravasated blood.

In the growth, both in its deep and superficial parts, are blood-vessels. Some of these are of large size, and appear to be merely channels in the new tissue, without any walls of their own. The growth is entirely free from pigmentation, and no areas of degeneration are visible.

This patient was readmitted on December 5, 1888. The growth had recurred "soon after" he left the Hospital in 1887, and had been slowly increasing in size since that date. The notes on admission (taken by Mr. Walker, the House-Surgeon) say: "*Right eye.* There is a growth about the size of a mulberry on the outer part of the cornea. It has a constricted base, and is flattened against the surface of the globe, from which it projects fully one-eighth of an inch. The inner edge of its widest part reaches to the mid-line of the cornea. It is firm to the feel, like gristle. Slight purulent conjunctival secretion. P. active. T. n. Cannot tell the time by a watch. No ophthalmoscopic changes."

The growth was removed by scissors, and was found to be pedunculated, its base about half as large as the area shadowed by the growth. The cornea beneath was slightly infiltrated. The base was touched with the galvano-cautery after removal.

The corneal wound healed kindly, and the patient left the Hospital in a few days.

The growth after removal resembled in shape the top of a mushroom, the concave surface showing traces of the peduncle by which it had been attached to the eyeball. It measured in its longest diameter 12 mm., and was thus more than double the size of the nodule removed in June, 1887. It was hardened in Müller's fluid, and sections made with a freezing microtome.

Microscopical Examination.—In its histological characters the recurrent differs in some details from the primary growth. There is no trace of epithelium on its surface, but a thin fibrous capsule surrounds it, from which strands of fibrous tissue penetrate and ramify through the cell structure, and give it an indistinctly lobulated arrangement. The cells are more generally round, although in many places long oval cells are evident. The vascularity of the tumour is greater than that of the former nodule, but the individual vessels appear smaller and

have better formed walls. There are no evident extravasations. As in the primary growth there is an entire absence of pigment.

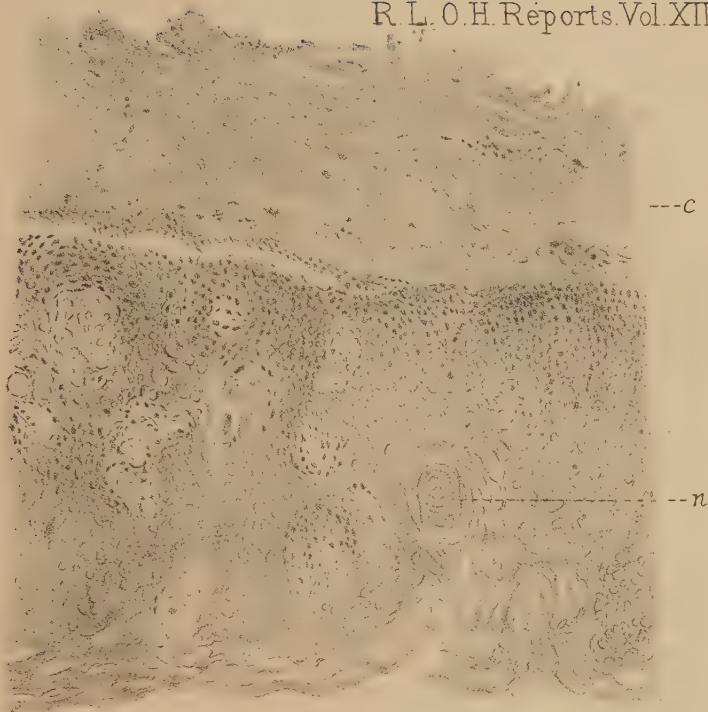
I am indebted to Mr. E. T. Collins, the present Curator of the Museum, for kindly handing over to me the recurrent growth.

DESCRIPTION OF PLATE.

FIG. 1 represents a section of the growth from Case 1. The fibrous part (*c*) is at the upper part, the cellular part below. The narrow space between these two parts at the left-hand side of the figure is artificial. $\times 160$.

FIG. 2 is a drawing of a section of the cornea from Case 3. Descemet's membrane is represented at *d*, and to the left of this are the posterior layers of the cornea, but little altered. These are succeeded by a narrow layer containing large numbers of small stained cells, and this layer by the epithelioma. At the lower end of the figure, the new growth is shown in about its greatest depth, and from this point a long extension of it (*e*) passes up in the small celled tissue, a portion of which has become enclosed between two layers of epithelial cell growth. $\times 120$.

FIG. 3 represents another portion of the same cornea, and shows the epithelial overgrowth dipping into the small cell layer beneath. $\times 70$.



A. Stone. del.

Fig. 1



Fig. 2

K. F. L. del.



Fig. 3

Danielsson & Co, lith.

PRIMARY SARCOMA OF THE IRIS.

By E. TREACHER COLLINS, *Curator of the Museum.*

ON August 12th, 1887, William Arthur H., aged 21, consulted me concerning a little brown spot at the outer and lower part of his left iris; he first noticed it three years ago, since then it has gradually increased in size, but he does not think that lately it has progressed more rapidly. His irides used both to be blue, the left has now become more of a green colour. He has never had any inflammation in the eye, nor has he at any time received any injury to it. His sight has always been good both for distant and near objects; the last few months, however, in the sunshine, with his right eye closed, he has observed a slight dimness; yesterday, for the first time, he noticed an appearance as of a circle of rainbow colours when he looked at the gas lamps. A photograph taken in February, 1883, shows a dark spot down and out on his left iris. His father and mother are living and well; none of his family as far as he is aware have ever suffered from any eye trouble or had tumours of any sort. His general health is quite good; he has never had syphilis or any serious illness. He is tall, robust, and of a fair complexion.

In his left eye some of the anterior perforating vessels are enlarged, especially those on the inner side; the cornea is clear; the pupil is active, but shows some irregularities in its margin at the lower part, probably posterior synechiæ. The iris is of a greenish colour mottled with brownish pigment; this pigmentation is most marked at the lower and outer part, and least at the upper and inner quadrant. Down and in the iris is swollen, and at the pupillary margin at this part is seen a rounded brown projection, the lens is clear, the optic disc and fundus are normal, T. full, V. = $\frac{6}{6}$ and J. 1.

In the right eye the pupil is active, the iris of a blue colour, no pigment spots on it, lens and fundus normal, V. = $\frac{6}{6}$ and J. 1. T. normal.

He was admitted into the Moorfields Hospital under the care of Mr. Nettleship, who has kindly permitted me to publish the case, and on August 17th, 1887, his left eye was excised.

On January 7th, 1889 (*i.e.*, nearly 1 year and 5 months since the operation), I heard from him, he was in good health, and there was no recurrence of the growth.

Pathological Examination.—The eye was hardened for three weeks in Müller's fluid, then frozen, and an antero-posterior oblique section made through the entire globe, running downwards and inwards; the structures were found to be all *in situ*, and (except the iris and ciliary body) showed no naked eye changes. The iris on the inner (nasal) side, and towards the lower part, was considerably thickened, the thickening being slightly irregular, and involving the whole width of the iris; the iris angle was nowhere blocked, but the iris and cornea were in contact. Near, but not quite at, the periphery there was patchy dark pigmentation of the iris over three-fourths of its extent. The ciliary body on the same side as the thickened iris was involved and considerably thickened. The lens, retina, choroid, and optic disc appeared normal.

Microscopical examination of sections through the thickened portion of the iris and ciliary body shows on the anterior surface of the iris a layer composed of closely packed small round cells, with very little intercellular substance, and only a small amount of cellular protoplasm surrounding a large deeply staining nucleus; this layer is thicker in some parts than in others, so that the surface of the iris is irregular. At the angle of the anterior chamber some of these round cells are seen lying between the layers of the cornea, and in and around the canal of Schlemm, and also lining for a short distance the posterior surface of

Descemet's membrane. Beneath this continuous layer of round cells are seen numerous small collections of similar cells, and some spindle-shaped cells extending throughout the substance of the iris, and separated from each other by bundles of unstripped muscular fibres and connective tissue; the ciliary muscle and processes are in like manner infiltrated with groups of round and spindle-shaped cells, the former everywhere predominating. There are a few scattered patches of pigment throughout the tumour; the blood vessels are not numerous.

Sections of the iris in the lower part, below the level of the pupil, show a somewhat different structure, represented in Figs. 2 and 3; the thickness of the iris here varies considerably in different parts, but nowhere is it so thick as in the other sections; the anterior layer is the most affected, but in places the whole thickness of the iris seems composed of small round cells. There are a large number of irregular masses of brown and black pigment, most marked in the anterior layer and around the blood vessels; pigment is also seen to be deposited as little granules in some of the cells. The blood vessels are numerous, and have thick muscular walls; the ciliary body on both sides is involved, but to a much greater extent on the inner side than the outer. On both sides a few cells are seen between the layers of the cornea, and also some in the sclerotic.

The literature of sarcoma of the iris has been worked up by Kipp, Knapp, and Little, so as to leave very little to be added. Galezowski states that he has only seen one case in 65,000 patients; 17 cases have been recorded in which the iris was the primary seat of the growth. To bring out some of the points in these cases I have tabulated them as follows:—

No.	By whom recorded.	Sex.	Age.	Nature of growth.	Situation of growth on iris.	Operation.
1	Le Brun	F.	36	Round celled, white.....	Started from the inner side near the ciliary margin	Iridectomy; only a portion of tumour removed; rapidity of growth increased. Excision.
2	Hirschberg	M.	38	Spindle celled, melanotic..	Lower part	Excision.
3	A. Robertson and H. Knapp	F.	24	Round celled, melanotic..	Largest growth at the upper and outer part, and extending downwards from this a chain of three other similar tumours	Excision.
4	Kipp.....	M.	36	Spindle celled, white.....	Inner and lower quadrant...	Iridectomy; 18 months, and no recurrence.
5	Knapp.....	M.	36	Spindle celled, white.....	No note.....	Iridectomy; 12 months, and no recurrence.
6	Knapp.....	F.	35	Spindle celled, melanotic..	Lower part	Iridectomy; three weeks later, eye quiet.
7	Knapp	M.	22	Round celled, white.....	Outer and lower part.....	Iridectomy.
8	St. John Roosa,...	F.	40	White	Inner side, ciliary margin...	Patient refused treatment.
9	Vose Solomon.....	F.	43	Spindle celled, melanotic..	Outer side ciliary margin; several secondary deposits	Excision.

No.	By whom recorded.	Sex.	Age.	Nature of growth.	Situation of growth on iris.	Operation.
10	Galezowski	F.	35	Melanotic	Lower part	Patient refused treatment.
11	Dreschfield	F.	53	Spindle celled, white.....	Lower part	Excision.
12	Romiée	F.	74	Round celled, melanotic ..	Outer part.....	Excision.
13	Carter	M.	15	Round celled, white.....	Both eyes affected: L., lower and inner quadrant; R., two small tumours	L., iridectomy; growth re- curred.
14	Adams	F.	13	Round celled, melanotic ..	Lower and outer part	Excision.
15	Little	F.	20	Round celled, melanotic ..	Outer and lower quadrant ..	Iridectomy; two years later no recurrence.
16	Von Hasner	..	..	Melanotic	Outer and upper part	Patient refused operation.
17	Hosch.....	M.	66	Spindle celled, melanotic..	Lower part	Excision.

Sex.—Out of the 18 cases (including my own) 10 were females, 7 males, 1 not stated.

Age.—2 between the ages of 10 and 20 years.

4	"	"	20	"	30	"
6	"	"	30	"	40	"
2	"	"	40	"	50	"
1	"	"	50	"	60	"
1	"	"	60	"	70	"
1	"	"	70	"	80	"

In one the age is not stated; the youngest case was aged 13, and the oldest 74.

Nature of Growth.—In 11 of the cases the growth was melanotic, in 5 of which round cells predominated, and in 4 the spindle-shaped cells; in 2 no microscopical examination had been made. In the cases recorded by Little and Hosch, and in my own, the pigment lay chiefly along the line of the vessels. In several of the cases, as well as in my own, a dark spot on the iris had been noticed for some time previous to the appearance of the neoplasm. Knapp and Hosch are of opinion that these dark spots from which the sarcoma starts are simple melanomata of the iris, which the former has described as a very rare affection, and similar to warts on the skin. It is interesting to note in connection with the multiple character of the tumours in some of the cases of sarcoma of the iris, that in one of the cases of melanoma recorded by Knapp, he states there were "a few small brown tumours."

In 7 cases the growth was unpigmented; 2 of these were composed chiefly of round cells, and 4 chiefly of spindle-shaped cells; in 1 no microscopical examination was made.

Situation of Growth on Iris.—The lower part of the iris was either partly or entirely the starting point of the growth in 11 cases, while only in 2 was the upper part affected, viz., Cases Nos. 3 and 16 in the table: in the former of these there was a chain of tumours extending downwards.

Knapp, in commenting on this case, discusses the question as to whether the single isolated tumours were "the result of a common morbid tendency in the iris, or the product of dissemination from the primary (larger) tumour." "The location of the larger tumour in the upper part of the iris," he says, "and the denser accumulation of the smaller tumours on the lower part, seem to support the dissemination theory." In my case there was also a chain of tumours, the largest one was at the lower and inner part, and lower than some of the secondary (smaller) nodules.

Operation.—Iridectomy was performed in seven of the cases. In Lebrun's case only a portion of the tumour was removed, and the operation seemed to hasten the rapidity of its growth; in one other case the growth recurred.

In none of those cases in which the eye has been enucleated was any recurrence recorded.

In the case I have related such a large extent of the iris was involved, that iridectomy was not attempted, and the pathological examination shows that it would have been useless, as the ciliary body was considerably involved, some of the sarcoma cells even extending into the neighbouring parts of the cornea and sclerotic.

To sum up, then, sarcoma of the iris is an extremely rare affection, occurring rather more frequently in females than males, most commonly between the ages of 20 and 40, but occasionally earlier or later in life. The growth may be melanotic or white, more often the former, in some of which occur secondary nodules, due probably to a common morbid tendency in the iris; it is composed of round and spindle-shaped cells, either of which may predominate and characterise the tumour; the lower part of the iris is the most frequently affected; as with other intraocular tumours it may give rise to glaucomatous symptoms. Iridectomy in many cases proves successful, but unless the whole of the growth is likely to be removed it is better to enucleate, as partial removal of the tumour stimulates the rapidity of the growth.

LITERATURE.

- Hirschberg, *Archiv. f. Ophthalmologie*, Bd. xiv, p. 285.
 Lebrun, *Annal. d'Oculist.*, vol. ix, p. 209, 1869.
 A. Robertson and Knapp, *Archives of Ophthalmology*, vol. iii, p. 106.
 Kipp, *Archives of Ophthalmology*, vol. v, p. 34.
 Knapp, *Archives of Ophthalmology*, vol. viii, p. 82.
 St. John Roosa, *Transact. American Ophth. Soc.*, 1869, p. 14.
 Vose Soloman, *Transact. of the Ophth. Soc. of the United Kingdom*, vol. ii, 1882, p. 257.
 Galezowski, *Recueil d'Ophthalmologie*, 1879, p. 727.
 Dreschfield, *Lancet*, Jan. 16th, 1875, p. 82.
 Romée, *Recueil d'Ophthalmologie*, 1831, p. 207.
 B. Carter, *Transact. Clinical Society*, vol. vii, 1874.
 Adams, *Lancet*, April 12th, 1879, p. 511.
 Little, *Transact. of the Ophth. Soc. of the United Kingdom*, vol. iii, 1883, p. 28.
 Von Hasner, *Prager Med. Wochenschrift*, S. 61 u. 62.
 Hosch, *Centralbl. f. prakt. Augenh. Leipz.*, 1881, p. 361.
 Knapp, *Treatise on Intra-Ocular Tumours*, pp. 289, 298.

DESCRIPTION OF PLATE.

FIG. 1 is from a drawing of the external appearance of the eye before operation. $\times 2$.

FIG. 3 represents a section through the lower part of the iris; and

FIG. 2 gives a highly magnified view of the part opposite *a* in Fig. 2.

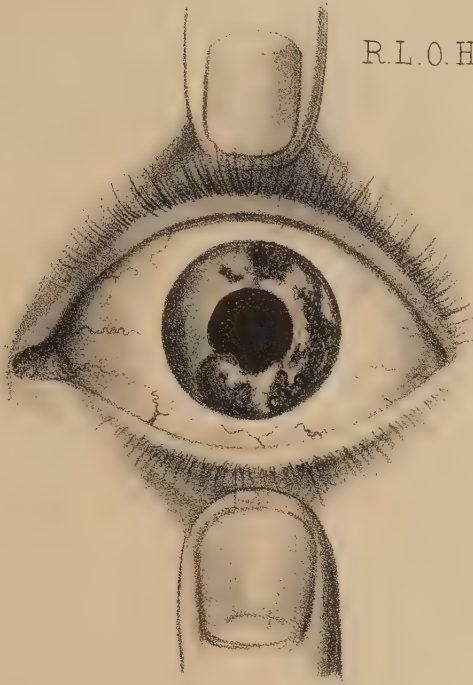


Fig 1.

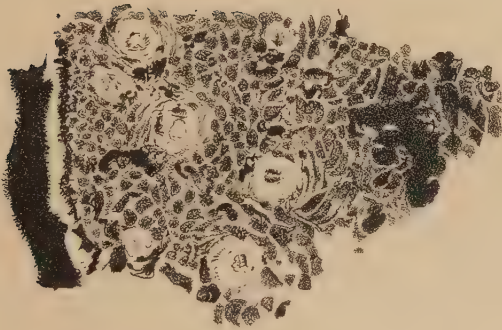


Fig 2.



Fig 3.

TWO CASES OF ORBITAL CELLULITIS, WITH NECROSIS OF
THE HORIZONTAL PLATE OF THE FRONTAL BONE,
ACCOMPANIED BY CEREBRAL ABSCESS.

By E. TREACHER COLLINS,
Curator of Museum; and

C. H. WALKER,
Senior House Surgeon.

CASE I.—John M., æt. 17, came to Mr. Tay as an out-patient at the Moorfields Hospital on July 14, 1887, stating that a month previously he had received a blow from a turnip on his left eye, after which the eyelids became swollen and very black. This had all passed away, but lately the upper lid had commenced to swell again. On examination it was found that there was an abscess pointing there, the skin over it being hot and discoloured. An incision was made into the centre of the lid, and some pus escaped; a drainage tube was then inserted. Two days later the swelling of the orbital tissues had considerably increased, the discharge became very foetid, and his temperature was 101° ; the left cornea was clear, and the eye and vision normal; the lids on the right side were œdematous. The incision on the left upper lid was then enlarged, to facilitate the free exit of pus, and a lotion containing perchloride of mercury used to irrigate the abscess cavity from time to time.

On July 18 (that is four days after the patient first presented himself at the hospital), at six o'clock in the evening, he had a rigor, and his temperature rose to 107° , after which he sweated freely; there was still considerable swelling of the orbital structures, and the wound was again enlarged towards the outer side of the orbit, iodoform powder being dusted in, and a lotion with chloride of zinc being substituted for the one with perchloride of mercury. During the next few days he had repeated rigors, his temperature at these times reaching 104° and 105° ; his tongue became red and dry; quinine in two-grain doses was administered every three hours.

On July 22 there was less swelling round the orbit, and less discharge, but on the 23rd a large fluctuating swelling appeared

over the left parietal bone; an incision was made into this, and a quantity of foetid pus made its escape; a chloride of zinc lotion was syringed in, iodoform dusted on, and a drainage tube inserted; on the evening of the same day an abscess appeared at the inner side of the nose, which was treated in a similar manner to that over the parietal bone.

On July 24, at 5 A.M., his temperature was 107° , pulse 122. Though he had had no rigors for two days, he vomited; at 10.30 A.M. he was barely conscious, lying with his head drawn to the left side, and throwing his arms about wildly. There was no change in the optic disc of the right eye; no ophthalmoscopic examination could be made of the left, on account of the swollen state of the lids. At 4 P.M. his temperature was 104° , and he was more comatose; he died at 6 P.M.

Post-mortem examination was made at 10 A.M., on July 25, 16 hours after death, by Mr. J. B. Lawford. Body fairly well nourished, little subcutaneous fat; no rigor mortis present; no effusion into joints. There is swelling and oedema of left eyelids, especially the upper; pus oozes from an incision beneath orbital rim at the outer angle, and through this bare bone can be felt in the roof of the orbit; there are two other incisions, one over the inner canthus in the upper lid, the other $1\frac{1}{2}''$ above the rim of the orbit. On removing the scalp there is suppuration beneath it over the left frontal and parietal bones, and the bone is exposed over an area $2'' \times 1\frac{1}{2}''$, on adjoining portions of temporal and parietal bones.

On removing the skull-cap the membranes are very adherent to the bone (especially over the left side), and thickened, yellow, and infiltrated; there is pus on the surface of the convolutions on both sides anteriorly; the superior longitudinal sinus is completely blocked anteriorly by dirty yellow clot. There is a circular opening, 4 mm. in diameter, with dirty ragged edges, in the anterior end of the second left frontal convolution, from which a thin, dirty, foetid pus exudes; this leads into an abscess cavity in the left frontal lobe, of irregular shape, the size of a large walnut, whose walls are of a greenish colour and ragged, with rapidly breaking down tissue adherent to them; it communicates with the anterior horn of the left lateral ventricle; its anterior, outer, and lower walls are very thin. The left lateral ventricle contains purulent fluid; in the right lateral

ventricle is some thinner sero-purulent fluid, and some yellowness along the edge of the choroid plexus. There is a soft grey patch on the anterior extremity of the left corpus striatum; otherwise no changes are found in the basal ganglia or their ventricle. At the base there is fairly widespread purulent meningitis; there is no perforation of the inferior surface of the left frontal lobe.

On the superior surface of the horizontal plate of the frontal bone on the left side there is a small patch of necrosis barely the size of a sixpenny piece. On removing the roof of the left orbit, a patch of bare necrotic bone is found in its anterior part the size of a halfpenny, its edge being just behind the orbital rim; beneath this, but apparently above the periosteum, is a small abscess cavity. There is pus in the frontal and ethmoidal sinuses; the left cavernous sinus is filled with semi-solid purulent material, but there is no suppuration in the superior petrosal or lateral sinuses of the left side.

The left optic nerve and the back of the eye removed; the optic disc is hazy, and the vessels hidden at its margins. The thoracic and abdominal viscera were not examined. Microscopical examination of sections of the left optic nerve near the eyeball and at the apex of the orbit show perineuritis and some increase of nuclei in the nerve, chiefly in the trabeculæ. In the anterior sections these changes are much less marked.

CASE II.—Bertha B., aged 15, was brought to the outpatient department on May the 11th, 1888, and placed under the care of Mr. Tweedy. She complained of a painful swelling over the left eye, which was said to have been first noticed three weeks previously, and to be getting worse. No history of injury was given at the time, nor was any cause assigned. There was considerable swelling of the left upper eyelid extending towards the bridge of the nose and upwards on the forehead. At a point corresponding to the inner end of the upper palpebral fold fluctuation could easily be felt; in fact, it was clear that there was an abscess almost pointing.

The patient was taken to the operating theatre, and whilst under an anæsthetic, an incision three-quarters of an inch long was made parallel to the inner end of the eyebrow, and about half an inch below it. About two drachms of thick pus were let out, and then the cavity was explored with a probe, and a

large area of bare bone at the inner side of the roof of the orbit was felt. The cavity was washed out with perchloride of mercury lotion, a drainage tube inserted, and the wound dressed with iodoform and antiseptic gauze. As a matter of precaution, Mr. Tweedy directed that the patient should be taken into the hospital and kept in bed.

The following day the swelling had much diminished, and the patient had no pain. Her temperature was normal. The wound was syringed and dressed in the same way as before. For the next two days there was nothing to notice except that the patient looked rather poorly and languid; she did not care to get up, and her appetite was bad. Her temperature was slightly raised, but was never above 100° F. There was very little discharge from the wound, which was syringed out daily and dressed with sal alembroth wool.

On May 18th (a week after the abscess was opened), the patient was distinctly worse. She was very drowsy, and complained of rather severe headache chiefly on the right side. She had taken scarcely any food for two days; her tongue was furred; bowels regular; pulse 100 to the minute, small and rapid; respiration shallow, about 30 to the minute; skin hot and dry, and cheeks flushed. The edges of the wound were pale, there was no discharge, and the drainage seemed to be quite free. No disease could be detected in the chest or abdomen. Ophthalmoscopic examination was made, but no changes were seen. Mr. Tweedy saw the patient, and ordered a dose of calomel to be given, and an ice bag to be kept applied to the head.

Shortly before midnight the patient seemed to be much the same; her temperature was 100°; she had been asleep some hours, and had not complained of anything.

The following morning (May 19th), after having a fairly good night, the patient was noticed to be rather more animated and lively than usual, but at 6:30 A.M. she again became drowsy. At 8:45 she suddenly became much worse. The house surgeon was called, and found her quite unconscious, and not able to be roused, except to move her limbs slightly. Her breathing was stertorous and her face congested. Heart's action violent, no murmur audible; pulse very irregular, 60 to 80 a minute. Temperature 99°·8. Pupils unequal, right 4 mm., left 7 mm.; no

changes seen in fundi. The wound was in the same condition as it had been the last three or four days. After a time, the limbs were seen to move in an irregular manner at various intervals. Excepting the dilatation of the left pupil there were no unilateral phenomena. No squinting or deviation of the eyes was observed. She continued in this state till 10 o'clock, when the breathing became much slower and more laboured, and the lips and face extremely congested. About six ounces of blood was drawn from the left arm, and this seemed to give slight relief, but it was only temporary. She died at 11.30 A.M.

The autopsy showed an irregular area of bare bone about the size of a sixpenny piece, some distance back in the roof of the orbit. On removing the upper portion of the cranium in the usual way, a quantity of pus escaped. This was found to come from an abscess situated in the left frontal lobe, about a quarter of an inch from the surface, and extending back into the left lateral sinus. The cavity, which was as large as a hen's egg, was filled with thin curdy pus of a pale muddy-red colour; there was no offensive smell. The walls of the abscess were covered with a slimy substance. There was no distinct membrane separating the abscess from the brain tissue. All round the cavity of the abscess the brain matter was soft and red, and the left corpus striatum was softer and distinctly smaller than the right. The convolutions of the brain were a little flattened and the veins distended. There was no pus or sign of inflammation in the other ventricles, and the rest of the brain substance seemed healthy. The membranes over the upper surface of the hemispheres were healthy, but at the base of the brain there were signs of meningitis, the pia mater being opaque, and the sub-arachnoid space filled with semi-purulent fluid. There were no signs of suppuration or thrombosis in the superior longitudinal or other venous sinuses. The dura mater was adherent to the left frontal lobe, and came away with the brain when the latter was removed, leaving the orbital plate of the frontal bone rough and carious. In the centre of this area, which was about an inch and a half long from before backwards, and an inch wide, there was a small hole, through which a good sized probe could be passed into the orbit and out through the external wound. In the dura mater there was a minute hole corresponding with this, but no distinct communication

with the abscess cavity in the brain could be detected. The disease had not affected the frontal or ethmoidal sinuses, nor were any other changes noticed elsewhere. The other organs were not examined.

These two very parallel cases present several points of interest. The apparently trivial nature of the injury in otherwise healthy individuals contrasts with the serious and fatal symptoms that followed. In Case II the only history of injury that could be obtained was, that the patient had a year previously run against some iron railings. This was not mentioned by the child's parents until after her death, and they were not at all certain as to the date or immediate consequences of the injury; at all events, the parents could not recollect the child being unconscious or laid up after it, nor could any scar of an external wound be found. There was no history of tubercle in the family, nor any symptom of it in the patient, and the only assignable cause of the abscess seems to be this trifling injury. In Case I the interval between the blow and the acute symptoms was not so long, being only a month; probably, also, the injury was more severe, as the patient was said to have had a "black eye" after it. Here again, however, there was no indication of there having been any external wound, nor could any other cause be discovered. In both cases the patients were young, being aged 17 and 15 years respectively, and in three similar cases recorded by J. Crawford Renton* and George S. Norton,† and quoted by Carter and Frost (from the *Archiv für Ophthalmologie*) in their treatise on "Ophthalmic Surgery," the ages were 12, 21, and 4 years.

The slightness of the symptoms presented when the patients first attended the hospital should, we think, engender great caution both in prognosis and treatment in all cases of abscess pointing in the eyelids. In Case II

* *Ophthalmic Review*, vol. v, p. 206.

† *Archives of Ophthalmology*, vol. xv, p. 30.

no symptom of any disease at all was complained of until the supra-orbital swelling was noticed a month before the termination of the case. There was no interference with any motor or sensory nerve (with the exception of the left pupil a few hours before death). There was no paralysis, fit, or spasm of any kind, and no impairment of the intellect until two days before the patient succumbed; there was no optic neuritis; headache was complained of, but it was of such a character that it might easily be accounted for by the patient's feverish condition. Excepting the hebetude noticed a few days before death there was no symptom to cause apprehension or indicate cerebral complication.

With regard to the course of the disease and the primary nature of the lesion, there are two possible explanations. First, that the injury caused an abscess of the orbit which set up periostitis (or primarily caused periostitis of the roof of the orbit) followed by necrosis and perforation of the bone, and subsequently by meningitis and abscess of the brain—in Case I the meningitis spreading upwards and backwards and causing a patch of necrosis in the parietal bone and the formation of an abscess beneath the scalp; for the abscess in the orbit and that over the parietal bone were apparently quite separate. Secondly, the injury may have caused an abscess of the brain, followed by meningitis, necrosis, and perforation of the roof of the orbit and an abscess pointing in the upper eyelid.

In favour of the first theory is the fact that in both cases the symptoms of disease in the orbit preceded any cerebral symptoms; though in Case II, as has been mentioned, the cerebral symptoms were never very definite. In Case I it was not until four days after the abscess in the lid (which had been forming for some days previous to his attendance at the Hospital) was opened that he had any rigor. Also, that periostitis from slight injuries about the age of puberty is of common occurrence.

In favour of the second view: in Case II the character

of the contents of the cerebral abscess, the atrophied condition of the neighbouring brain matter (especially the corpus striatum) and the fact that the dura mater in one place was very adherent, would all lead one to suppose that the abscess must have existed a considerable time. The position, also, of the necrosed bone, namely, in the horizontal plate of the frontal bone in both cases, seems an unlikely one to have been affected by a trifling blow without any perforating wound.

With regard to treatment, there is one point of practical interest which is suggested by these cases, and that is with reference to drainage of the orbit, especially when the abscess is situated as far back as it was in both these cases. In Case I the incision was originally made in the middle line, and it was found that the boy had to be frequently turned on his face to allow of any pus escaping by the tube. Subsequently the wound was enlarged outwards, and then by turning on his left side free drainage was possible. We would suggest that in all cases where it is evident that an abscess in the orbit has to be opened the incision should, if possible, be made on the outer side.